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BENIGN TUMORS OF THE LARYNX

A STUDY OF SEVEN HUNDRED AND TWENTY-TWO CASES

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Benign tumors of the larynx are of interest and importance to the laryngologist, not only because of the symptoms which they produce by interference with the normal functions of the vocal mechanism or by obstruction of the respiratory tract but because of the necessity of distinguishing them from malignant laryngeal lesions. Tumors of a bengin nature do not occur frequently in the larynx. During the past thirty years 722 patients with benign laryngeal growths have been examined at the Mayo Clinic. It is interesting to find that these tumors, apparently, are encountered less often than are malignant neoplasms, as approximately 1,100 malignant tumors of the larynx were observed during this same period.

In any discussion of benign tumors of the larynx, there arises the need of defining the term "benign laryngeal tumor." Occasionally, there occur benign new growths, composed of cells which resemble various normal tissue cells but which fulfill no physiologic function; such growths as these may be termed correctly "true benign neoplasms." More frequently in the larynx there develop inflammatory swellings or hyperplastic growths, both of which originate as physiologic responses to some form of irritation; although the word "tumor" is applied loosely to growths of this type, nevertheless they are not true tumors. Since by clinical methods it is not always possible to distinguish a true benign laryngeal neoplasm from an inflammatory or a hyperplastic overgrowth, one must interpret any abnormal mass of tissue in the larynx as a benign tumor, providing that it lacks the infiltrative qualities and the power to metastasize characteristic of a malignant lesion. Such a broad interpretation embraces a wide variety of laryngeal conditions: true proliferative neoplasms; nonproliferative tumors, as

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represented by retention cysts; growths of a hemorrhagic or of an inflammatory origin, and those of a hyperplastic nature. Although not included among the benign laryngeal tumors, specific inflammatory growths, such as tuberculoma, syphiloma and blastomycosis, require careful consideration.

No classification of benign laryngeal tumors is altogether satisfactory. Clinically, it is an impossibility to distinguish many of these tumors, because they bear such close resemblance one to another. The histogenesis of some is so obscure that a pathologic classification cannot be prepared with complete accuracy. Consequently, in consideration of the present extent of knowledge, a classification is neither practical from a clinical standpoint nor entirely possible from a pathologic point of view.

TABLE 1.—*Histologic Classification of Benign Tumors of the Larynx*

	Number	Percentage
Neoplasms (329, or 45.6 per cent)		
Tumors of epithelial origin		
Adenomas	1	0.1
Papillomas	194	26.9
Tumors of connective tissue origin		
Fibromas	6	0.8
Neurofibromas	1	0.1
Fibrolipomas	1	0.1
Chondromas and osteochondromas	7	1.0
Angiomas	26	3.6
Myxomas	58	8.0
Cysts	35	4.8
Non-neoplastic tumors (393, or 54.4 per cent)		
Inflammatory tumors	332	46.0
Xanthomas	4	0.6
Amyloid tumors	18	2.5
Epithelial hyperplasia and leukoplakia	33	4.6
Prolapse of the ventricle	6	0.8
Total	722	99.9

However, some sort of systematic grouping is necessary for studying the various tumors under discussion. Analysis of our 722 cases has led us to adopt the classification shown in table 1, which is based on the histologic appearance of the tumors. Using the term "neoplasm" to imply a true tumor and the word "tumor" itself to denote any abnormal mass of tissue, we have attempted to separate benign laryngeal growths into two primary groups: neoplastic and non-neoplastic tumors. The former group has been subdivided into three: tumors of epithelial origin, tumors derived from connective tissue elements and cysts. The latter group consists of a composite group of tumors, none of which is a true neoplasm and most of which are inflammatory. Although leukoplakia cannot be considered a tumor, it frequently appears similar to benign tumor of the larynx. Prolapse of the ventricle is intimately associated with an inflammatory process which closely resembles a common type of pedunculated laryngeal tumor. Therefore, leukoplakia and prolapse of the ventricle have been included in our list of tumors.

Many inflammatory tumors undergo cellular changes which both clinical and histologic methods fail to distinguish from those of true neoplasms. This difficulty in distinguishing true neoplasms from pseudoneoplasms is strikingly apparent in the myxomatous tumors, some of which are true neoplasms and others the result of an inflammatory process. Since it is difficult to decide which growths are true myxomas and which are pseudomyxomas, we have decided for convenience to classify all myxomatous tumors arbitrarily as true neoplasms. The same applies to the angiomas, many of which probably are vascular inflammatory growths. True congenital cysts are known to occur in the larynx, but those which we have seen were all retention or hemorrhagic cysts; perhaps these should not be included among the true neoplasms. No doubt, if their histogenesis were better understood, many lipomas and chondromas would not be classified as true tumors. If these uncertainties and probabilities are borne in mind, it is evident that our grouping of these tumors is not a classification at all. As MacCallum [1] put it, in discussing his classification of tumors, "It is an arrangement, not a classification."

It would be a matter of interest to know how many of our 722 tumors were actually true neoplasms. To make such a determination would be not merely difficult but impossible, since it is likely that many of the growths which we have classified as neoplastic are not true tumors. Papillomas, of course, are more frequently encountered than any other true benign neoplasms and form 27 per cent of our total group of tumors. They differ, however, in many respects from other benign tumors of the larynx. The only tumors in our 722 cases, aside from papillomas, that we could call true neoplasms were a few myxomas, a few angiomas, 7 chondromas, 6 fibromas, 1 neurofibroma, 1 fibrolipoma and 1 adenoma. As previously stated, possibly even some of these growths were not true neoplasms. At any rate, it is evident that a true benign laryngeal neoplasm (excluding papillomas) is uncommon.

ETIOLOGY

Although many hypotheses have been advanced to explain the formation of benign tumors, their actual cause is still unknown. In the larynx probably the great majority of benign tumors are the result of a chronic inflammatory process. Regions of chronic inflammation in this organ may be due to irritation, overuse or misuse of the voice or any of the innumerable causes of chronic laryngitis. Frequently a submucous hemorrhage is the etiologic factor in producing a region of chronic inflammation. Several of our patients gave a history of becoming

1. MacCallum, W. G.: A Text-Book of Pathology, ed. 4, Philadelphia, W. B. Saunders Company, 1928.

suddenly hoarse after a period of shouting. Examination revealed a hemorrhagic nodule on a vocal cord, usually at the junction of the anterior and the middle third.

Hemorrhagic and simple inflammatory areas may disappear, but frequently the inflammatory process does not subside; instead there is a proliferation and piling up of the connective tissue elements, producing tumor-like formations. These inflammatory growths in some instances become very vascular and then can be easily confused with angioma. Some undergo fibrosis and occasionally produce a fibrous tumor that is difficult to distinguish from true fibroma. Many of the inflammatory tumors that we have examined contained regions of hyalinization. In others regions of myxomatous degeneration were to be found; when a large portion of the tumor has undergone myxomatous changes, to determine whether it is merely inflammatory or a true myxoma is more than perplexing.

Inflammation is the basic factor in the development of many benign tumors. According to one accredited theory, the formation of some

TABLE 2.—*Age of Patients in 722 Cases of Benign Tumor of the Larynx*

Age of Patients	Cases, Percentage
11 weeks to 25 years	18.4*
26 to 50 years	59.8
51 to 76 years	21.8

* In 79 per cent of these cases the tumors were papillomas.

lipomas in the larynx takes place by fatty infiltration in a region of chronic inflammation. Probably chronic inflammation is an important agent in the development of all xanthomas. No doubt, too, a chronic inflammatory process is necessary for the formation of amyloid tumors.

INCIDENCE

Although benign tumors of the larynx can occur at any age, they are most frequently encountered in middle-aged persons; 59.8 per cent of our patients were between the ages of 25 and 50, and 21.8 per cent were between the ages of 50 and 76 (table 2). Only 18.4 per cent were below the age of 26, and 79 per cent of these patients were children who had multiple papillomas. Thirty-five to 50 is the age at which these tumors most commonly develop.

Our youngest patient was a child of 11 weeks, who had a laryngeal angioma associated with multiple angiomas of the body. Our oldest patient was a man of 76, who had an inflammatory nodule on the anterior third of the left vocal cord.

Benign laryngeal tumors afflict men more frequently than women. Five hundred and six, or approximately 70 per cent, of our 722 patients were men; 216, or 30 per cent, were women.

PATHOLOGY

Benign tumors are composed of well differentiated cells, which displace rather than infiltrate tissues. They never metastasize. In the larynx they produce symptoms by compressing the adjacent tissues, by mechanically interfering with the vocal mechanism or by obstructing the respiratory tract.

Although there are wide variations, in general the histologic appearance of true neoplasms is that of normal tissue. In inflammatory tumors there is present the cellular picture of chronic inflammation, in which the connective tissue elements predominate. True neoplasms never are of any physiologic value to the rest of the body, whereas an inflammatory tumor originates as a physiologic response to some form of irritation.

The benign tumors of our group varied in diameter from less than 1 mm. to proportions commensurate with the size of the entire laryngeal lumen. Most of them measured 3 to 5 mm. in diameter.

Multiple, Different Tumors.—Occasionally two or more distinct and different tumors are present simultaneously in the larynx. We have had 10 such cases:

Case 1.—A man aged 62 had a subglottic adenoma attached to the anterior two thirds of the right vocal cord and a myxomatous polyp attached to the entire left vocal cord.

Case 2.—A woman aged 39 had a pedunculated polypoid tumor attached to the posterior third of the right vocal cord and a contact ulcer granuloma at the left vocal angle.

Case 3.—A man aged 49 had polypoid thickening of the anterior third of the right vocal cord and prolapse of the left ventricle.

Case 4.—A man aged 27 had a fibrous inflammatory nodule on the anterior third of the left vocal cord and epithelial hyperplasia of the anterior third of the left false cord.

Case 5.—A man aged 66 had a fibromyxomatous polyp at the base of the left vocal cord, an inflammatory polypoid nodule at the anterior commissure and an inflammatory tumor with degenerating subepithelial tissue on the upper surface of the epiglottis.

Case 6.—A woman aged 21 had a papilloma on the posterior third of the right vocal cord and a fibrous polypoid nodule on the posterior third of the left vocal cord.

Case 7.—A woman aged 50 had bilateral amyloid nodules at the junction of the anterior and middle thirds of the vocal cords and an inflammatory nodule with epithelial hyperplasia at the base of the left vocal cord.

Case 8.—A man aged 52 had epithelial hyperplasia of the anterior two thirds of the right vocal cord and multiple papillomas at the anterior commissure.

Case 9.—A woman aged 51 had an epithelial hyperplastic nodule at the junction of the anterior and the middle third of the right vocal cord and an inflammatory nodule at the junction of the anterior and the middle third of the left vocal cord.

CASE 10.—A man aged 42 had a cyst at the junction of the anterior and the middle third of the right vocal cord and an inflammatory nodule on the middle third of the left vocal cord.

Adenomas.—The histologic appearance of an adenoma is well known. It is a tumor composed of tubules or acini of cuboid or columnar epithelial cells surrounded by a vascular stroma. Adenomas of the larynx are true neoplasms, are usually polypoid or pedunculated and originate in connection with the mucous glands. Since these tumors have no ducts through which the contents of the acini can be expelled, they frequently become cystic. The one adenoma which we have had occasion to examine contained a large mucous cyst (fig. 1). This tumor

Fig. 1.—Adenoma containing a mucous cyst (× 50).

was pedunculated, measured 1.5 cm. in diameter and was attached to the under surface of the anterior two thirds of the right vocal cord.

Papillomas.—A papilloma is a papillary neoplasm covered with epithelium and having a core of vascular connective tissue (fig. 2). Developing in any part of the larynx, papillomas repullulate on the surface and do not invade the submucosa. Usually they are multiple and in children often fill the entire larynx. Of our 194 patients who had papillomas, only 28 had a single papilloma, and all these were adults. In adults multiple papillomas are often grouped together to form a lobulated or mulberry-shaped tumor. Although these tumors are benign, they have a marked tendency to recur after removal. Rarely does a papilloma undergo malignant change; in 3 of the 194 cases, squamous

cell epithelioma of which the malignancy was of grade 1 was discovered in a mass of papillomas (fig. 3). Removal of the papillomas resulted in cure in all these cases.

Fibromas.—A fibroma is a tumor composed of tissue which resembles fibrillar connective tissue (fig. 4). The 6 fibromas which we have seen at the clinic were soft pedunculated tumors, consisting of young loose areolar tissue and numerous blood vessels, and were infiltrated with wandering cells. They ranged from 2.5 mm. to 2.5 cm. in diameter.

Fig. 2.—Papilloma of the larynx (× 50).

Fibromas are true neoplasms and are not to be confused with inflammatory tumors that have undergone fibrosis; the latter growths occur frequently in the larynx, while true fibromas are rather rare.

Neurofibromas.—Neurofibromas arise either from the sheaths of nerves or from nerve roots. In the former instance they are known as "peripheral neurofibromas" and in the latter as "central neurofibromas." In the larynx they are of the peripheral type and are a great rarity. At the clinic but 1 such tumor has been seen. It measured 3 by 8 mm. and was attached to the middle third of the right vocal cord.

Strictly speaking, these growths are not connective tissue tumors, as they arise from the cells composing the sheath of Schwann, which

are of ectodermal origin. However, connective tissue does enter into the formation of these tumors.

Microscopically, as described by Figi,[1a]

The outstanding histologic features are the palisade-like and parallel arrangement of the nuclei, which are usually elongated. The nuclei also tend to be

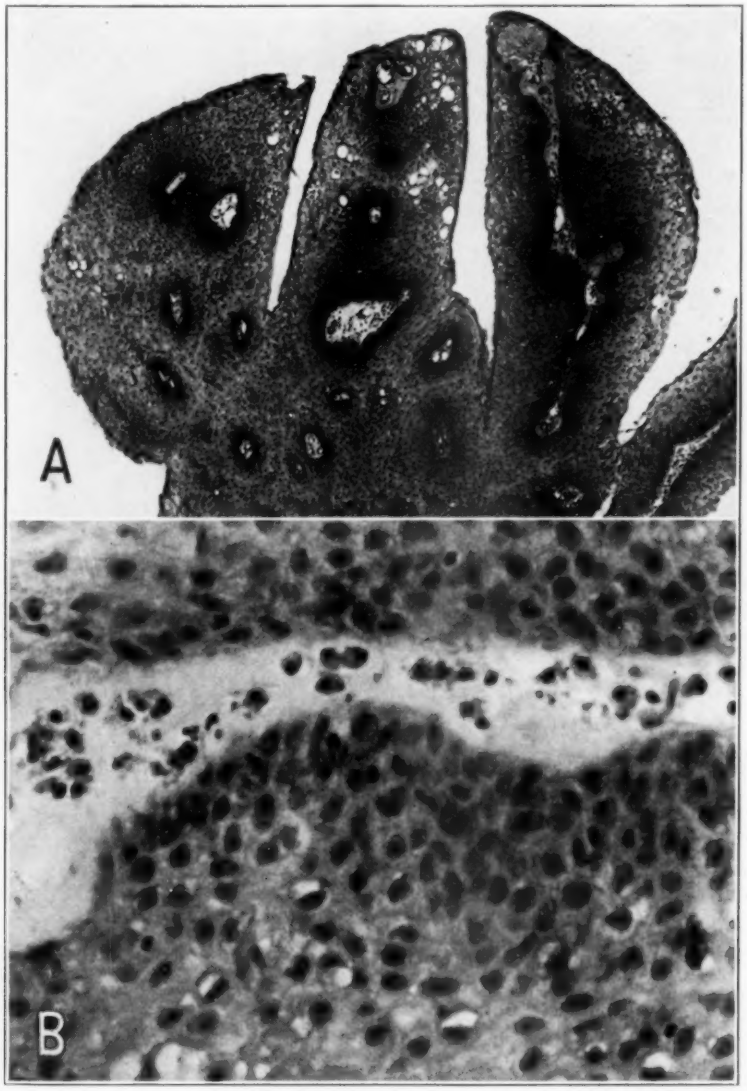

Fig. 3.—*A*, papilloma containing a grade 1 squamous cell epithelioma in situ (× 50). *B*, increased magnification of a section in *A*, showing mitotic figures (× 350).

1a. Figi, F. A.: Solitary Neurofibroma of the Pharynx, Arch. Otolaryng. **17:** 386-389 (March) 1933.

arranged in streams and eddies. Running parallel to the long axis of the nuclei are long, parallel fibers of collagen. Blood pigment is often found scattered through the tissues. Another microscopic feature that some observers have considered characteristic of such tumors is the presence of "foam cells." These are large, vacuolated, phagocytic cells present in the degenerated areas. Small fluid-filled cysts also occur in the degenerated portions of the tumor (fig. 5).

Fibrolipomas.—Lipomas are neoplasms composed of fat cells and some fibrous tissue. When the connective tissue elements predominate, they are termed "fibrolipomas" (fig. 6). Both lipomas and fibrolipomas are rare tumors in the larynx. They may be small polypoid pedunculated tumors or may be situated deep in and expand the tissues of the larynx. The 1 fibrolipoma seen at the clinic was of the latter type

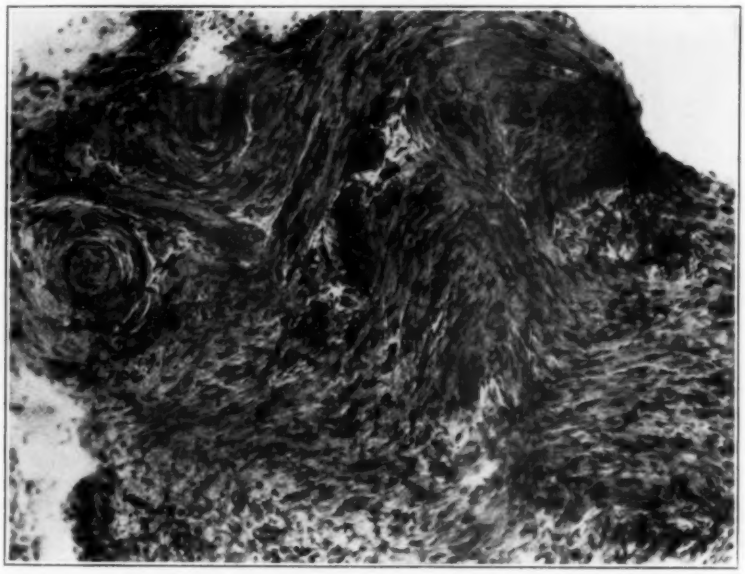

Fig. 4.—Fibroma of the larynx (× 140).

being located in the left arytenoepiglottic fold. The gross specimen was rounded and measured 2.5 cm. in diameter. Microscopically, it contained mostly fibrous tissue, with some fat cells at the periphery; the fatty tissue increased toward the center of the tumor. There were many regions of degeneration in this growth, which was diagnosed histologically as a "degenerating fibrolipoma."

Chondromas and Osteochondromas.—True chondromas probably arise from fetal cartilaginous rests and are rather uncommon in the larynx. As stated by one of us,[2] Alexander [3] has classified chondromas

2. New, G. B.: Cartilaginous Tumors of the Larynx, Laryngoscope **28**: 367-375 (May) 1918.

3. Alexander, A., cited by New.[2]

of the larynx as follows: (1) chondromas which are true neoplasms; (2) chondromas of mixed tumor type (chondromyxomas, chondrosarcomas and so forth); (3) inflammatory chondromas; (4) chondromas characterized by general hypertrophy of one or all of the cartilages of the larynx. We have seen none of the latter two types and have not included mixed tumors in our list of benign laryngeal growths.

Histologically, chondromas consist of hyaline cartilage (fig. 7 *A*). Ossification occurs in some of them (fig. 7 *B*) and calcification in others.

Fig. 5.—Neurofibroma, showing palisade-like and parallel arrangement of nuclei; "foam cells" are present (× 100).

Alexander stated that there is irregularity in the size, shape and arrangement of the cells, which show unusual energy of growth. In the older tumors, a cyst may form from a retrogressive change in the tumor. Bell [4] stated that a chondroma may undergo metaplasia and become myxomatous (myxochondroma); other pathologists would call such a growth a mixed tumor.

4. Bell, E. T.: Textbook of Pathology, Philadelphia, Lea & Febiger, 1930.

The 8 chondromas in our group of cases were all typical microscopically. One contained regions of ossification (osteochondroma).

Angiomas.—True angiomas of the larynx are not common neoplasms. A great many laryngeal tumors diagnosed as angiomas are pseudoangiomas, that is, varices, lymphectases or vascular inflammatory tumors. True angiomas may be hemangiomas or lymphangiomas. Histologically, hemangiomas are either simple, consisting of newly formed blood vessels in a stroma of connective tissue (fig. 8*A*), or cavernous, composed of large intercommunicating blood spaces surrounded by fibrous connective tissue. Normal blood is found in these

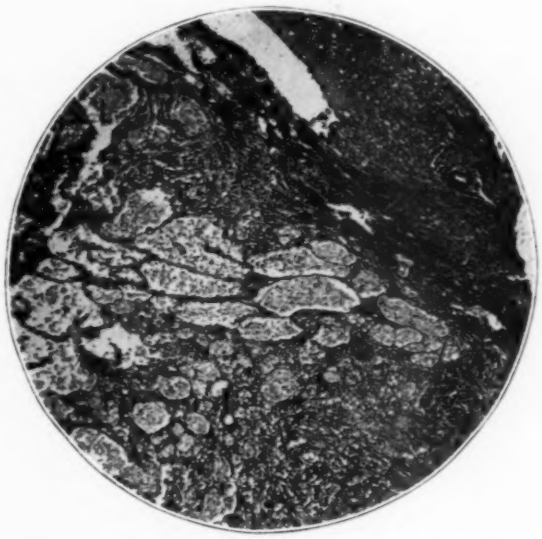

Fig. 6.—Fibrolipoma, showing islands of fat cells interposed between fibrous connective tissue (× 50).

capillaries and blood spaces. Lymphangiomas consist of dilated lymph spaces lined with endothelium and contain a fluid resembling true lymph (fig. 8*B*).

The cavernous hemangiomas are encountered more frequently than the simple types and vary in size from 2 to 2.5 cm. usually. Occasionally they are pedunculated, but more often they are sessile on a broad base.

Many simple inflammatory growths undergo vascularization, and it is exceedingly difficult, if not impossible, to distinguish some of these pseudoangiomas from true angiomas in which inflammatory cellular elements are present.

Twenty-five angiomas have been seen at the clinic. This number does not include those vascular tumors that were definitely diagnosed as pseudoangiomas on an inflammatory basis. Furthermore, we believe

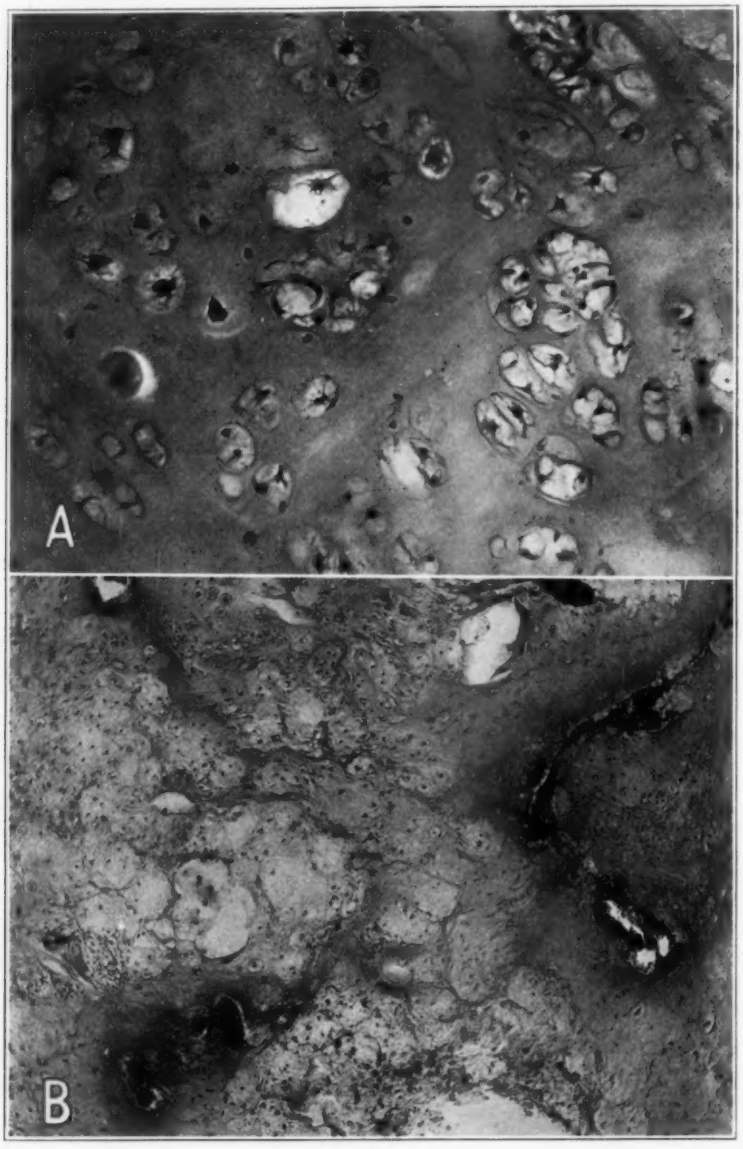

Fig. 7.—*A*, chondroma of the larynx (× 160). *B*, osteochondroma, showing centers of ossification in a chondroma (× 60).

that a few of these 25 angiomas were of inflammatory origin. Only 1 of the 25 tumors was a lymphangioma; the rest were hemangiomas.

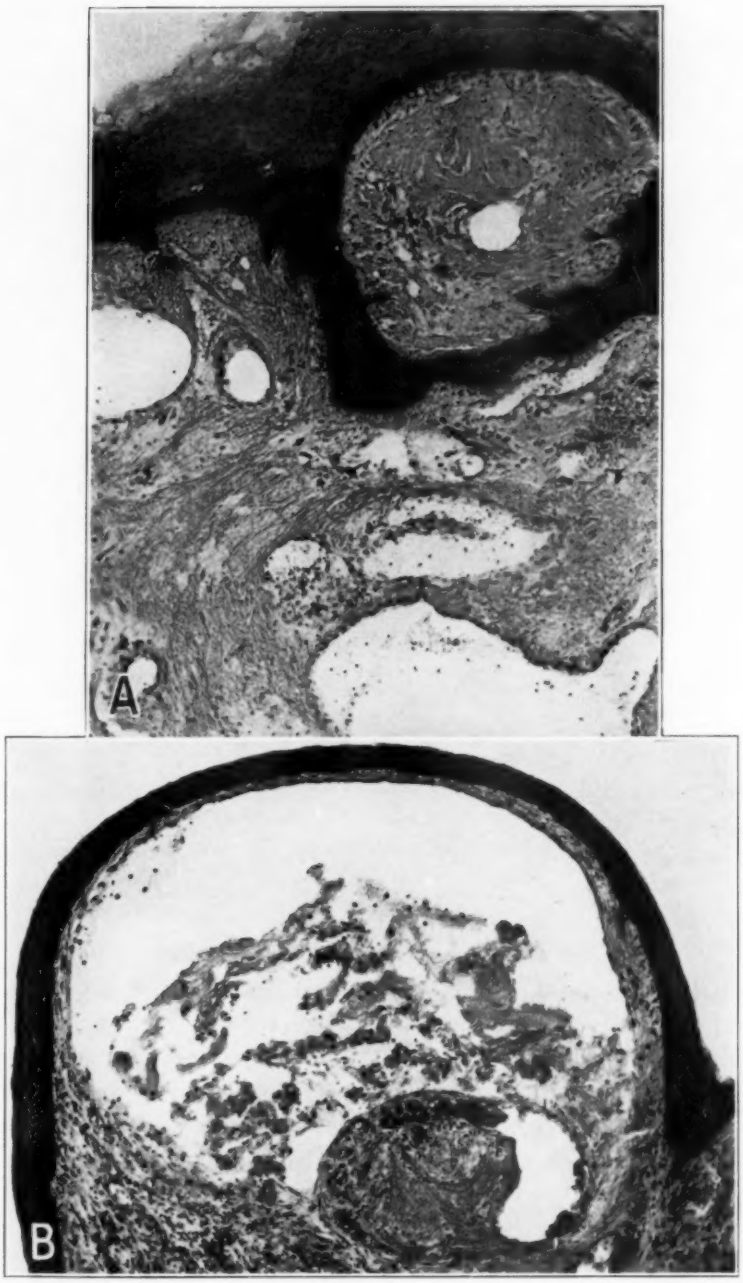

Fig. 8.—*A*, hemangioma of the larynx (× 65). *B*, lymphangioma covered with squamous epithelium (× 55).

Myxomas.—Myxomatous tissue is found normally only in the umbilical cord, but it resembles closely the tissue found in myxomatous tumors.

Myxomas are composed of mucous tissue, a modified form of connective tissue. Microscopically, they contain loose connective tissue, with branched stellate cells in a matrix of viscid mucoid material. The fibrous tissue may predominate, forming a fibromyxoma (fig. 9). In the larynx they are well defined tumors and usually polypoid.

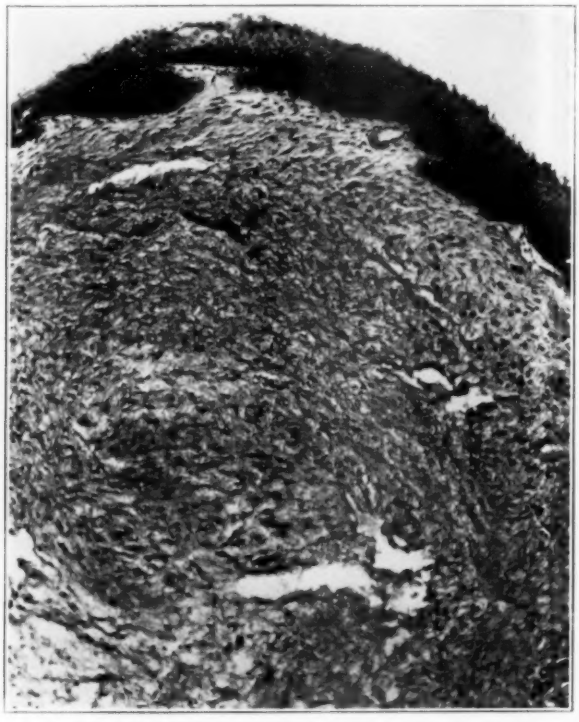

Fig. 9.—Fibromyxoma covered with squamous epithelium (× 50).

Myxomatous tumors and polyps are seen frequently, but few are true neoplasms, most of them being of inflammatory origin. In an inflammatory tumor the connective tissue elements may acquire a myxomatous appearance. This is termed "myxomatous degeneration" by some pathologists, as the condition is associated with signs of degeneration in the cells. However, Bell interpreted a myxomatous change in a tumor as attributable to metaplasia rather than to a degenerative process. He stated that any connective tissue tumor (osseous, cartilaginous, adipose, areolar) may undergo metaplasia and become myxomatous.

The various myxomatous tumors seen at the clinic are listed in table 3 according to their histologic appearance.

Cyst.—There are three types of laryngeal cysts: (1) mucous cysts, which are formed by obstruction of the duct and accumulation of mucoid secretion in a mucous gland; (2) hemorrhagic cysts, which are formed in submucous extravasations of blood or lymph; (3) embryonal cysts, which are congenital and which may be present at birth or develop later in life. Several hypotheses, reviewed by one of us,[5] have been offered to explain the formation of these tumors. Schneider[6] expressed the belief that they arise from embryonal cells sequestrated from the cells which form the appendix of the ventricle. The opinion expressed by Louys[7] was that they are derived from mesodermic tissue of the branchial arches.

TABLE 3.—*Myxomatous Tumors*

Tumors	Number
Myxomas	3
Fibromyxomas	6
Adenomyxomatous polyps	1
Fibromyxomatous polyps	3
Hemorrhagic fibromyxomatous polyps	1
Inflammatory fibromyxomatous polyps	1
Myxomatous polyps	29
Hemorrhagic myxomatous polyps	2
Myxomatous polyps with hyaline degeneration	1
Vascular myxomatous polyps	4
Inflammatory myxomatous polyps	7
Total	58

Mucous cysts are encountered more frequently than other types of laryngeal cysts (fig. 10). At first they are lined with columnar epithelium, but this epithelial lining may gradually disappear, owing to the pressure of the fluid within the cyst.

Hemorrhagic cysts have no epithelial lining but are filled with coagulated blood or with a serous fluid containing varying amounts of fibrin.

Embryonal cysts are rare tumors. They have an epithelial lining, and there are numerous glands in the wall of the cyst. Schneider reported an embryonic cyst the diagnosis of which was made from the presence of numerous glands and much elastic tissue in the wall of the cyst in which were small lymph follicles.

Difficulty in making a microscopic diagnosis of laryngeal cyst is often encountered, because the lining of these cysts is frequently absent. Moreover, cysts often collapse in the course of their removal, leaving only a small piece of tissue for histologic study.

5. New, G. B.: Cysts of the Larynx, Journal-Lancet **37**:99-102 (Feb. 15) 1917.

6. Schneider, P., cited by New.[5]

7. Louys, E., cited by New.[5]

Of the 35 cysts in our group of benign laryngeal tumors, 1 embryonal cyst was found involving the right aryepiglottic fold, and only 1 was diagnosed as a hemorrhagic cyst on microscopic examination.

Fig. 10.—*A*, mucous cyst lined with columnar epithelium and covered with squamous epithelium (× 65). *B*, mucous cyst in which a portion of the lining has become atrophied (× 50).

Clinically, however, several of them appeared to be hemorrhagic. The remaining cysts were of mucous type.

Inflammatory Tumors.—Inflammatory tumors are the most common of all benign laryngeal growths. We have seen 332 tumors that were definitely inflammatory (table 4). On microscopic section (table 5), 40 per cent showed simple inflammatory tissue (fig. 11 *A*); 20 per cent were hemorrhagic (fig. 11 *B*); 2 per cent were vascular (fig. 12 *A*); 37 per cent were fibrous (fig. 12 *B*), and 1 per cent were granulomatous (fig. 13 *A*). Areas of hyaline degeneration were present in 10 per cent of these tumors (fig. 13 *B*).

These types of inflammatory tumor represent various phases of cellular activity which occur in an inflammatory process. Whatever irritant induces the development of an inflammatory process also stimu-

TABLE 4.—*Clinical Classification of Inflammatory Tumors of the Larynx*

Type of Tumor	Number	Percentage
Fibrous nodules	96	28.9
Polypoid tumors	168	50.6
Pedunculated tumors	11	3.3
Papillary tumors	17	5.1
Inflammatory thickenings	21	6.3
Contact ulcer granulomas	19	5.7
Total	332	

TABLE 5.—*Histologic Classification of Inflammatory Tumors of the Larynx*

Type of Tissue	Percentage
Simple inflammatory tissue	40
Hemorrhagic inflammatory tissue	20
Vascular inflammatory tissue	2
Fibrous inflammatory tissue	37
Granulomatous tissue (nonspecific)	1

Ten per cent showed hyaline degeneration.

lates the growth of cells. Endothelial cells may proliferate to form capillaries and vascularize the tumor. In some cases the fibroblasts are activated, and areolar connective tissue is formed and fibrosis produced. In an inflammatory region hemorrhages occur frequently. Some laryngeal tumors begin as a submucous hemorrhage, which later may undergo inflammatory changes. The development of a granuloma in or around a contact ulcer represent's nature's attempt to heal the ulcer. Of little significance in a tumor are areas of hyalinization, which betray a degenerative process, that is, chemical changes in necrotic and coagulated cells.

Fibrous inflammatory tumors are not fibromas; the former are never circumscribed or encapsulated as are true fibromas. Rather, they show on microscopic examination only subepithelial fibrous tissue in a chronic inflammatory mass. Fibrosis is not present in a newly formed inflammatory tumor but develops as the inflammatory process becomes chronic.

Various chemical and cellular activities in regions of chronic inflammation probably account for the formation of xanthomas, amyloid tumors, some lipomas and chondromas and many myxomatous tumors.

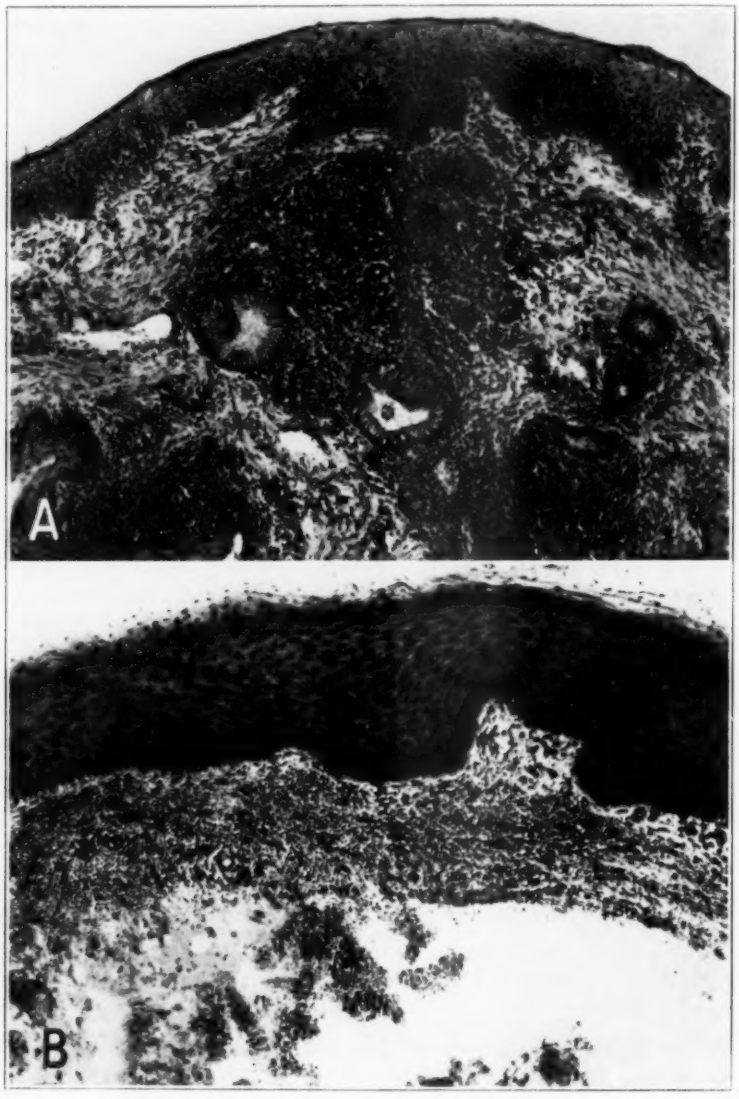

Fig. 11.—*A*, simple inflammatory tumor (× 90). *B*, hemorrhagic inflammatory tumor (× 125).

(Small patches of myxomatous degeneration were observed in many of our tumors.)

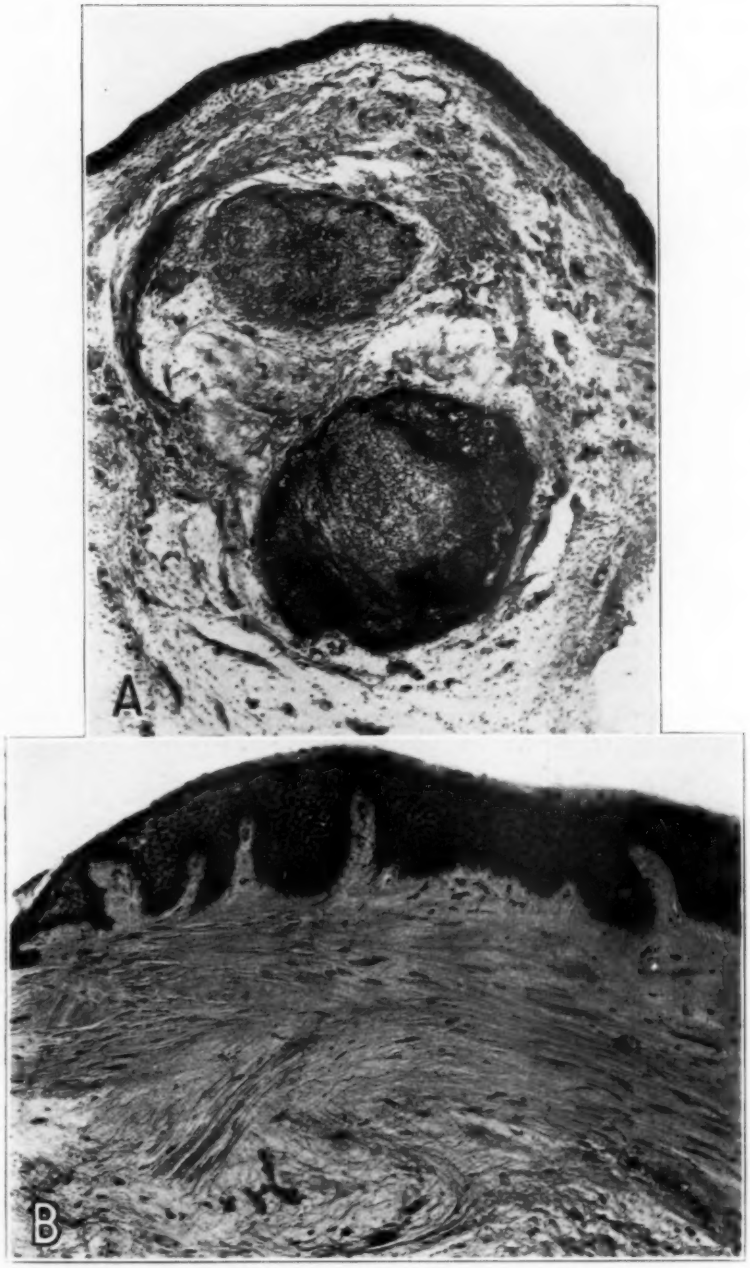

Fig. 12.—*A*, vascular inflammatory tumor (pseudoangioma) containing two thrombosed vessels (× 50). *B*, inflammatory tumor with subepithelial fibrosis (pseudofibroma) (× 90).

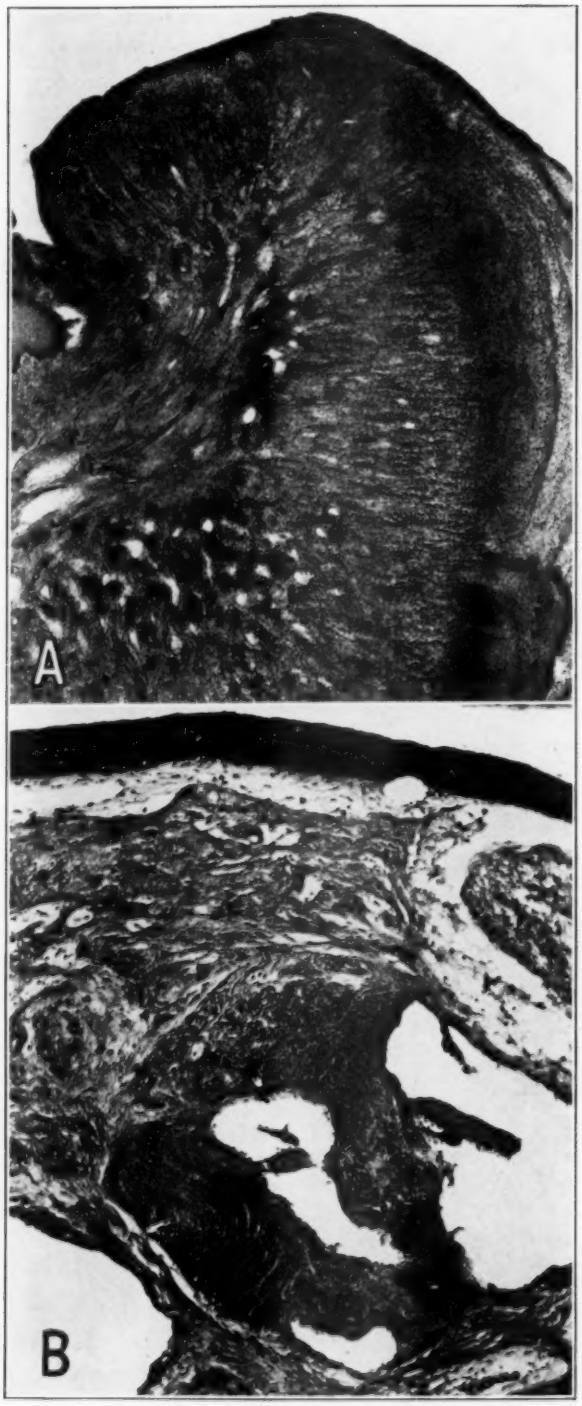

Fig. 13.—*A*, contact ulcer granuloma (× 20). *B*, inflammatory tumor undergoing hyalinization (× 50).

Clinically, inflammatory tumors appear as fibrous nodules, polypoid tumors, pedunculated tumors, inflammatory thickenings, papillary growths and contact ulcer granulomas (fig. 2).

Xanthomas.—Xanthomatous lesions are uncommonly encountered in the larynx. They are frequently seen around the eyelids and elsewhere on the skin, in muscles and in joints. In the mouth, pharynx, larynx and trachea they occur as nodules or plaques; they usually are multiple, have a smooth or nodular surface and possess a yellowish color.

Xanthomas undergo various phases of evolution, which have been arbitrarily divided into three stages: (1) a beginning inflammatory stage, (2) an intermediate tumor stage and (3) a final stage of fibrosis, which represents a regressive process. Histologically, they show a stroma of connective tissue, in which lie many cells of various sizes. The cells most diagnostic are the large "foam cells," so called because of the accumulation of lipoid droplets in their cytoplasm, which produces a foamy appearance. Other cells noticeably present, particularly in the tumor stage, are "Touton" cells, which are multinucleated giant cells. Their nuclei are centrally arranged in a circular fashion, and their cytoplasm contains the same fat droplets as are present in foam cells (fig. 14).

In 9 of the 16 cases of xanthoma in which quantitative chemical analysis of the blood was performed, the concentration of cholesterol and lipoids in the plasma was increased. It is commonly stated in the literature that patients with xanthomas have hypercholesteremia. This would appear likely, since xanthomatosis frequently accompanies such conditions as diabetes mellitus, nephritis, pregnancy or jaundice, all of which are associated with hypercholesteremia.

The foam cells are thought to be derived from the reticuloendothelial system. They are normal large phagocytic cells, which are attracted to regions of inflammation. In patients with hypercholesteremia these cells become laden with cholesterol esters derived from the blood stream. The globules of cholesterol esters and a lipochrome in the foam cells give the yellow color to the tumor.

Three requirements are then necessary for the development of a xanthoma: an area of inflammation, histiocytes (phagocytic cells) and hypercholesteremia. The activity of histiocytes in a region of inflammation in a patient with hypercholesteremia may explain the development of many xanthomas, but other factors must enter into the formation of some xanthomatous lesions, as hypercholesteremia is not always present in cases of xanthomatosis.

Four cases of xanthoma of the larynx have been observed at the clinic. Strangely, in 3 cases there was no increase in the concentration of cholesterol in the plasma; in the fourth case the values for cholesterol and lipoids in the plasma were not determined.

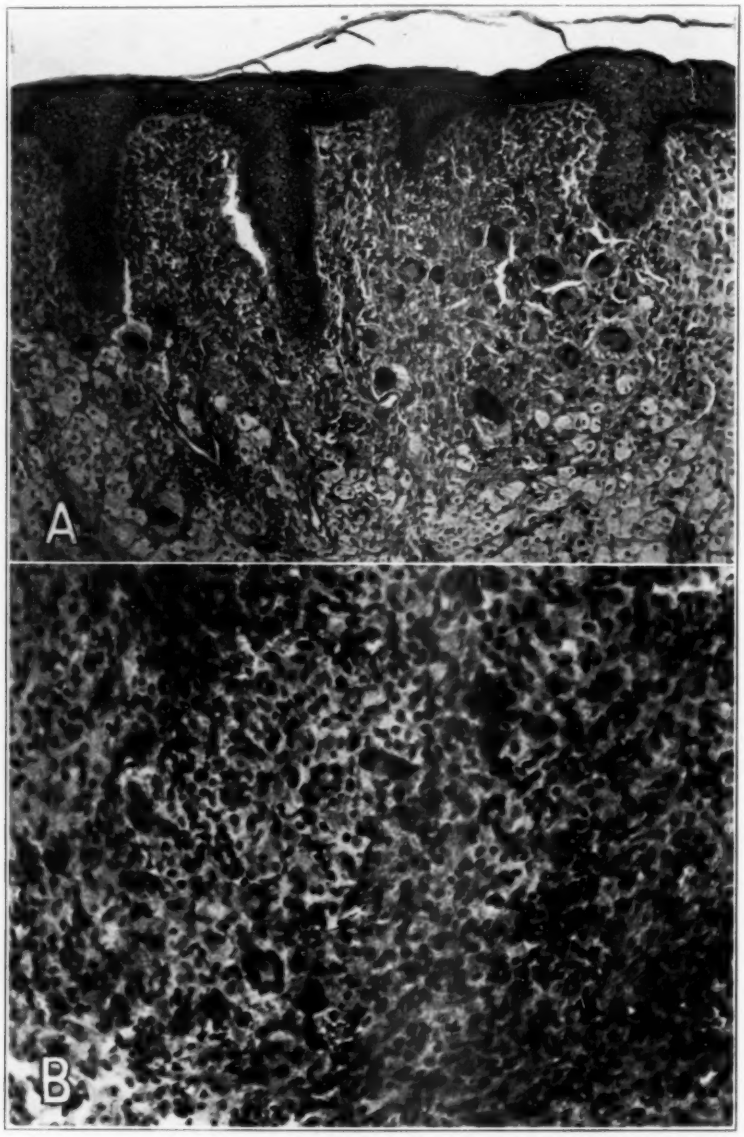

Fig. 14.—*A*, xanthoma, showing connective tissue stroma and "foam cells" (× 75). *B*, increased magnification of a section in *A* (× 205).

In 2 cases xanthomas of the larynx were associated with multiple xanthomatous lesions about the body and with diabetes insipidus. In 1

case xanthomas of the larynx were associated with several other xanthomatous lesions of the body. In this case the patient had pernicious anemia, a disease in which the concentration of cholesterol in the plasma is always low. In the remaining case a nodular xanthoma 2.5 cm. in diameter involved the posterior two thirds of the true and false vocal cords on the left side. No other xanthomatous lesions were found in this case.

The association of diabetes insipidus with xanthomatosis is thought to be due to mechanical interference with the normal functioning of the pituitary body, owing to the formation of a xanthoma in or adjacent to the pituitary body.

Amyloid Tumors.—Amyloid tumors of the larynx are not true neoplasms, as they are not formed by cellular growth but by the deposition of amyloid substance in the crevices of the tissues of localized regions in the body.

Amyloid is a firm translucent material, the chemical composition of which varies greatly in different tumors. Its chemical composition is still uncertain; where and how it is formed are still matters of conjecture. However, it is certain that all amyloid contains chondroitin-sulfuric acid, which is found normally in cartilage. Although amyloid may be a product of cellular activity, it is never found within the cells of any tissue but occurs between the cells. It is an infiltrate.

General amyloidosis is found, especially in the abdominal organs, in cases of chronic suppurative or destructive disease. Tumor-like local deposits occur most often in or near tissues containing cartilage, which probably accounts for the fact that amyloid tumors are found more frequently in the tissues of the upper part of the respiratory tract than in any other part of the body. In the cases of amyloid tumor which we have observed, there was no chronic wasting disease, although in some there were general pathologic conditions which might account for the development of amyloid.

Amyloid tumors of the larynx arise from three conditions: (1) diffuse subepithelial infiltration, (2) a local deposit of amyloid and (3) amyloid degeneration of a preexisting tumor. The term "amyloid degeneration," although generally used, is misleading, as the process is infiltrative rather than degenerative.

As described by New,[8]

> the microscopic pathology of this condition depends on the type of tumor present. In the diffuse, and in the tumor forming type of local amyloidosis, a subepithelial infiltration of the amyloid substance is noted. The blood vessels are especially affected. The mucosa and the submucosa are elevated by the amyloid deposit which is seen in layers closely packed between the connective tissue.

8. New, G. B.: Amyloid Tumors of the Upper Air Passages, Laryngoscope **29**:327-341 (June) 1919.

In the case of amyloid degeneration of a tumor, . . . the characteristics of the original growth as well as of the amyloid degeneration may be found. The amyloid gives the characteristic staining reaction with iodin, and shows up distinctly with hematoxylin and eosin (fig. 15).

At the clinic we have seen 18 amyloid tumors. Five were of the diffuse subepithelial infiltrative type; 4 were localized tumors, like deposits of amyloid, and 9 were inflammatory tumors which had undergone amyloid degeneration.

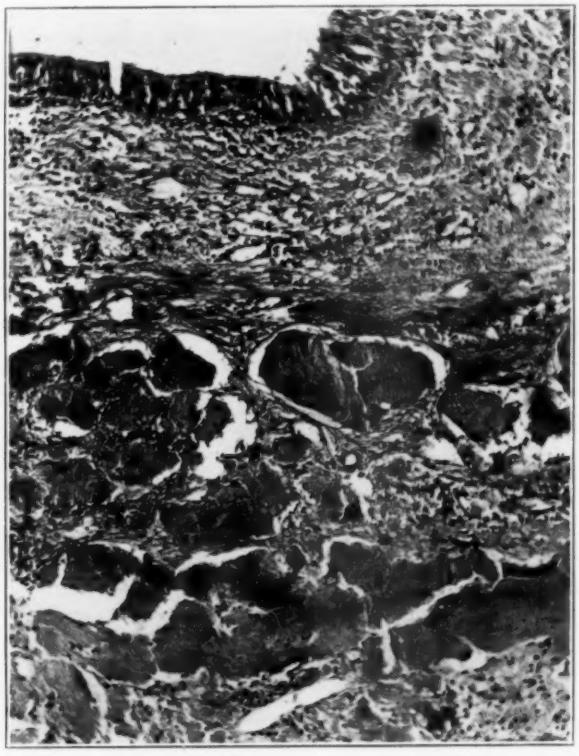

Fig. 15.—Amyloid tumor of the larynx (× 100).

Epithelial Hyperplasia and Leukoplakia.—Prolonged irritation in the larynx may result in hyperplastic epithelium, leukoplakia or both.

Epithelial hyperplasia, often termed "keratosis" of the larynx, forms whitish masses of piled-up epithelial cells (fig. 16*A*). Hyperplastic epithelium is frequently encountered in the epithelium, covering other benign tumors of the larynx. Leukoplakia, as is well known, appears as a white membrane and consists of hyperkeratosis of the epithelial cells in a much thickened mucous membrane with epithelial downgrowths (fig. 16*B*). In both leukoplakia and epithelial hyperplasia there are marked subepithelial inflammatory changes. These conditions have been considered together because they are intimately related to

cancer. This is particularly true of leukoplakia (fig. 17), although epithelial hyperplasia is also potentially malignant. Epithelial hyperplasia is more frequently seen in the larynx than is leukoplakia. We have encountered 23 cases of the former and 10 of the latter condition.

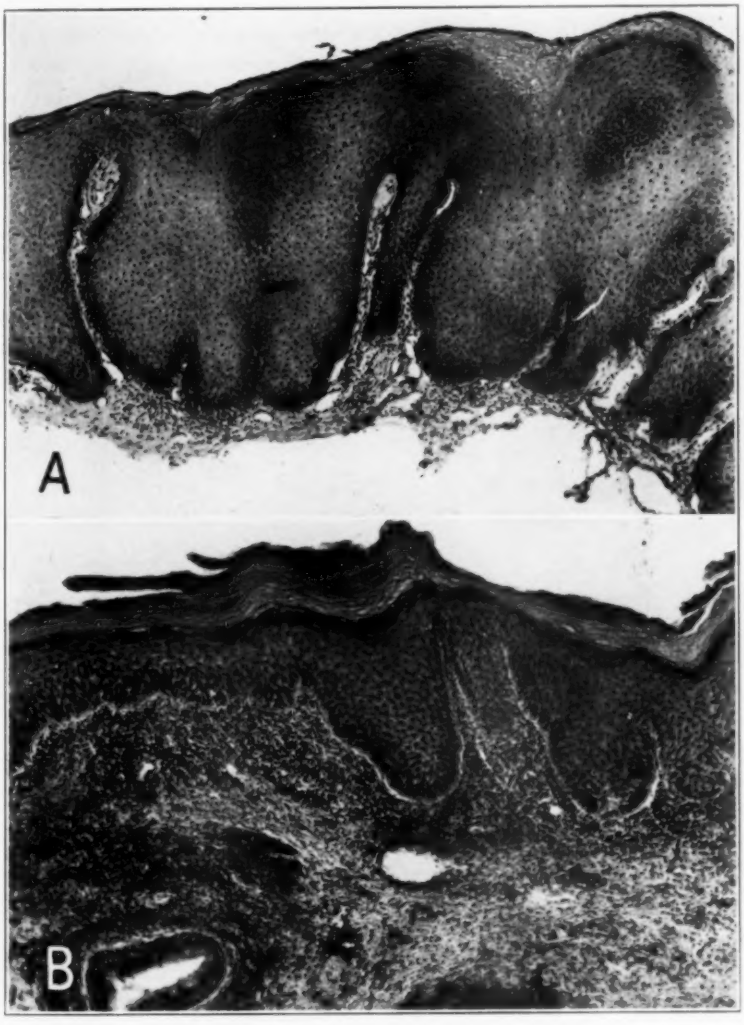

Fig. 16.—*A*, epithelial hyperplasia (× 50). *B*, leukoplakia of the larynx (× 80).

Cases in which leukoplakia was definitely associated with an epithelioma were not included. However, in 2 of the 10 cases a squamous cell epithelioma, grade 2, in situ, was discovered histologically; this emphasizes the importance of thorough microscopic examinations in these two conditions.

Prolapse of the Ventricle of the Larynx.—A prolapse of the ventricle of the larynx consists of a displacement of the mucous membrane of the laryngeal ventricle in which the mucosa protrudes into the lumen of the larynx between the true and the false vocal cords.

Moore [9] has thoroughly reviewed the literature concerning prolapse of the ventricle. Many causes have been assigned to explain this condition (local relaxation of the ventricular mucous membrane, actual invagination of the mucous membrane, coughing, syphilis, tuberculosis,

Fig. 17.—Leukoplakia containing a squamous cell epithelioma, grade 2 (× 90).

submucous cysts or neoplasms and edematous hyperplasia of the subepithelial connective tissue).

At the clinic 6 cases of prolapse of the ventricle have been observed; in 3 cases the prolapse was on the right side, and in 2 cases it was on the left side; in 1 case the condition was bilateral. In 1 case the patient declined operation, so that no tissue was available for microscopic study.

9. Moore, I.: Prolapse of the Laryngeal Ventricle, and Eversion of the Sacculus, in Jackson, C., and Coates, G. M.: The Nose, Throat, and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, pp. 907-925.

In the remaining 5 cases the involved tissue was removed, but histologic examination showed nothing but marked inflammatory tissue and granulations (fig. 18).

SITUATION

The site of predilection for benign tumors of the larynx is the vocal cords. A study of our group of tumors (excluding multiple papillomas, which were scattered throughout the larynx) showed that the tumors

Fig. 18.—Prolapse of the laryngeal ventricle, showing marked inflammatory changes in the subepithelial tissues; mucous glands are apparent in the lower portion of the section (× 50).

were ten times more frequent on the vocal cords than in all other parts of the larynx. Tumors occurred thirty-one times more frequently on the true than on the false cords, the second most common site. It was found that the interarytenoid region was involved least frequently; only 2 tumors were situated in this region. Even in the cases of multiple papillomas, the papillomas arising on the vocal cords far outnumbered those occurring in all other parts of the larynx.

A tumor may be attached to the free edge or the upper or lower surface of a vocal cord, or it may lie in the substance and produce expansion of the cord. Five hundred and fifty-five tumors were situated on the vocal cords; 292 occurred on the right side, and 246 occurred on the left side; 17 were situated in the anterior commissure. Eight per cent involved the entire length of the cords; 28 per cent, the anterior third (the most common site); 21 per cent, the middle third; 8 per cent, the posterior third; 6.5 per cent, the anterior two thirds, and 0.5 per cent, the posterior two thirds; 25 per cent arose at the junction of the anterior and the middle third of the vocal cords (table 6).

TABLE 6.—*Situation of Benign Laryngeal Tumors Attached to the Vocal Cords**

Situation of Tumor	Cases, Percentage
Anterior commissure	3.0
Entire length of cords	8.0
Anterior third	28.0
Middle third	21.0
Posterior third	8.0
Anterior two thirds	6.5
Posterior two thirds	0.5
Junction of anterior and middle third	25.0

* Ninety-two per cent of all benign tumors (excluding multiple papillomas) were attached to the vocal cords.

TABLE 7.—*Symptoms of Benign Tumors of the Larynx*

Symptoms	Cases	
	Number	Percentage
Hoarseness	687	95.2
Dyspnea	79	10.9
Cough	48	6.6
Pain or soreness	24	3.3
Sensation of fulness	10	1.4
Hemoptysis	4	0.6
Dysphagia	3	0.4
None	32	4.4

SYMPTOMS

There are no symptoms characteristic or diagnostic of benign tumors of the larynx. In fact, in 32 cases the patient did not complain of any laryngeal symptoms whatsoever (table 7). In these cases the growths either were small or were situated in regions other than the vocal cords, and there was no interference with phonation. The tumors in these cases were discovered during examination of the ear, nose and throat or during preoperative examination of the vocal cords, which is a routine procedure at the clinic in all cases in which thyroidectomy is performed.

Hoarseness is the most constant symptom. Including the 32 cases in which there were no laryngeal symptoms, hoarseness was absent in only 35 cases. A tumor in the larynx produces hoarseness mechanically, by direct or indirect interference with the normal movements of the

vocal cords. There may be marked variations in the degree of hoarseness; it may be slight or severe or intermittent or persistent. In some cases it amounts to nothing more than a slight huskiness of voice and a desire to clear the throat on arising in the morning. Frequently a patient with a benign laryngeal tumor says that he is able to phonate clearly in the morning—but hoarseness soon develops and increases during the day; by night his voice is reduced to a mere whisper. Although hoarseness may develop suddenly in a case of benign laryngeal tumor, in the majority of our cases the onset was insidious, and there was a gradual increase in severity.

The most serious symptom produced by benign laryngeal growths is labored breathing or dyspnea, which should receive careful consideration. Most of the tumors are not of sufficient size to produce any apparent shortness of breath, and even with the larger growths the dyspnea is usually not alarming. Benign tumors of the larynx, whether single or multiple, may be of considerable size but may produce surprisingly little respiratory difficulty. However, with the accumulation of tenacious mucus between the tumor and the laryngeal wall, a severe choking spell may ensue; if the mucus is not dislodged or if the dyspnea is not relieved, death may result from complete asphyxiation. Several of our patients, especially those with multiple papillomas, gave a history of persistent mild respiratory obstruction and occasional attacks of violent choking. Such conditions are potentially dangerous and should receive prompt attention.

In severe dyspnea all the accessory respiratory muscles are brought into play in the patient's effort to obtain sufficient air. Usually there is stridor, which may be inspiratory, expiratory or both. Cyanosis is present and gradually deepens.

Multiple papillomas produce dyspnea more frequently than do other benign tumors. In 20 cases in which there were multiple papillomas, the patient had only a slight or moderate amount of dyspnea when first seen at the clinic. In 25 other cases of multiple papillomas, there was marked respiratory difficulty when the patient came to the clinic. In 3 of these cases the obstruction was so severe that the patient was almost asphyxiated. Dyspnea was present in only 28 of the 528 cases in which tumors other than multiple papillomas were present, and in 9 of these cases the shortness of breath was either entirely functional or was not due to laryngeal obstruction.

In evaluating any symptoms, the functional element must always be taken into consideration. Many patients who have neurotic tendencies and are aware of the presence of a laryngeal tumor can imagine all sorts of symptoms relative to the pharynx or larynx. This is particularly true of patients who complain of pain or a feeling of a lump in the hypopharynx. In 24 of our cases the patient said he had suffered

from pain or soreness in the throat, but in 16 cases there was absolutely no cause of such discomfort. In the remaining 8 cases there was contact ulcer granuloma or another inflammatory lesion over a vocal process. In 10 cases the patient complained of a sense of fulness, a "lump" in the throat; in 5 of these cases the cause was entirely functional, and in each of the other 5 cases a large tumor was attached to the epiglottis or to the tissues of the upper laryngeal aperture.

Coughing is not an outstanding symptom of benign laryngeal tumors. Only about 7 per cent of our patients complained of cough.

In 4 cases there had been attacks of hemoptysis, and in 3 cases there had been difficulty in swallowing. In 1 of the latter cases the dysphagia was functional.

In the entire series of cases the duration of symptoms previous to examination varied from two days to forty years. In 11 cases the patient said that he had had symptoms from twenty to forty years. However, in some of these cases, it seemed doubtful that a laryngeal

TABLE 8.—*Duration of Symptoms in 722 Cases of Benign Tumor of the Larynx*

Duration of Symptoms	Cases, Percentage
2 days to 3 months	12
4 to 11 months	23
1 to 2 years	30
2 to 5 years	18
6 to 10 years	7
11 to 20 years	8
21 to 40 years	2

tumor had been present for such a long time. The largest number of patients had had symptoms from one to two years, as is shown in table 8.

DIAGNOSIS

The symptoms which have been enumerated are not diagnostic of benign laryngeal tumors or any other condition in the larynx. These symptoms are as characteristic of laryngitis, syphilomas, tuberculomas, malignant neoplasms and other laryngeal lesions as they are of benign tumors. Any patient with symptoms referable to the larynx, particularly hoarseness and dyspnea, should be subjected to thorough laryngoscopic examination.

The gross appearance of many benign tumors and other laryngeal lesions, such as epitheliomas, tuberculomas and syphilomas, may be so similar that in many cases it is impossible to differentiate these conditions by clinical examination alone. In such cases the assistance of laboratory tests, a general physical examination and in some instances biopsy are required before a correct diagnosis can be assured.

At the clinic every patient with laryngeal symptoms is required to give a thorough history of such symptoms. Roentgenograms of the

thorax, a complete blood count, urinalysis, serologic tests for syphilis and examination of the sputum, if indicated, are made, and a general physical examination is performed. The laryngoscopic examination is carried out by the indirect method. When a laryngeal tumor is found, if it appears perfectly benign and if it is accessible, the entire growth is removed under suspension laryngoscopy and sent for microscopic examination. A report of the histologic examination of a frozen section is obtained before the patient is dismissed from the operating room. In cases in which tumors are questionably malignant or in which a diagnosis cannot be made by observation, a specimen for biopsy may be obtained with indirect laryngoscopic vision before any treatment is undertaken. At the clinic no piece of tissue which has been removed from the larynx is ever discarded until a thorough microscopic examination has been made.

Differential Diagnosis of Benign Tumors of the Larynx.—Benign tumors of the larynx must be distinguished from malignant tumors, tuberculomas, syphilomas, acromegaly of the larynx, blastomycosis and torulosis.

Malignant Tumors of the Larynx: Early laryngeal epitheliomas of a projecting papillary character that have not infiltrated deeply enough to cause fixation of the vocal cord may be confused with benign papillomas. However, the latter appear more papillary and, as we have found, are usually multiple; carcinomas are single lesions in the larynx. The base of the tumor, commonly congested when the lesion is malignant, is associated with little or no inflammatory reaction when it is benign. Papillary inflammatory growths are often more difficult to distinguish from papillary carcinomas than are papillomas, and the diagnosis frequently can be accomplished only by biopsy. A clinical differentiation between a papillary leukoplakia and a similar-appearing epithelioma is unnecessary, since all thickened leukoplakia should be considered malignant until proved otherwise by microscopic examination. An occasional early infiltrative carcinoma without ulceration may simulate one of many types of benign tumor, but when the infiltration becomes more extensive and affects the normal mobility of the vocal cords the diagnosis offers little difficulty. Ulceration is extremely uncommon in benign tumors, but it occurs frequently in malignant lesions. Malignant ulceration usually presents an excavated surface, with irregular everted edges, and it is surrounded by a zone of deeply congested tissue. In benign tumors, pain is a rare symptom, unless the lesion is situated over a vocal process. As a symptom of a malignant laryngeal neoplasm, pain may be constant, but it is not present until late in the course of the disease, at which time the diagnosis of the lesion is perfectly obvious. When carcinoma of the

larynx becomes fungating or undergoes ulceration, when the vocal cord becomes definitely fixed or when a metastatic mass develops or metastatic involvement of the lymph nodes of the neck occurs, there can be little question as to the nature of the condition. In all cases in which the diagnosis is doubtful it can and should be precisely determined by removal of a piece of the growth for microscopic examination.

Tuberculosis: Laryngeal tuberculosis frequently involves the interarytenoid region, which we have found to be an extremely uncommon situation for the development of a benign tumor. The mucous membrane of the larynx usually maintains a normal appearance and color in the presence of a benign tumor. In tuberculosis, after an initial stage of hyperemia, the mucosa becomes pale, in contrast to the usual appearance of the mucous membrane of the larynx with benign tumors and to the marked congestion with syphilis and malignant lesions. When the tuberculous process involves the epiglottis and the arytenoepiglottic folds, as it frequently does, the patient may complain of extreme pain on swallowing. Pear-shaped swellings of the aryteno-epiglottic folds are diagnostic of tuberculosis. Tuberculous ulceration is usually superficial, with irregular inactive margins. Occasionally laryngeal tuberculosis may acquire a papillary appearance, which may simulate benign papillomas; the latter, however, are more warty and cauliflower-like. Moreover, tuberculous papillary lesions are frequently associated with other manifestations of tuberculosis, such as edema, paleness of the mucosa and ulceration. The discovery of active pulmonary tuberculosis and of tubercle bacilli in the sputum, together with the clinical laryngeal findings, usually offers sufficient evidence to justify making a diagnosis of tuberculosis of the larynx. When tubercle bacilli are absent from the sputum, animal inoculation should be tried. One should not overlook the possibility of a benign or malignant neoplasm of the larynx in the presence of active pulmonary tuberculosis; in some cases only biopsy will establish a correct diagnosis. Spencer [10] has given a thorough account of tuberculosis of the larynx.

Syphilis: Syphilitic lesions of the larynx are more often confused with malignant than with benign tumors. A laryngeal syphiloma is usually associated with hyperemia of the mucous membrane, and the infiltrative stage develops rather rapidly. Ulceration, when present, is deep and has a punched-out appearance and undermined edges. Pain is present when the process involves the epiglottis. The Wassermann reaction of the serum and that of the spinal fluid are positive, and the

10. Spencer, F. R.: Laryngeal Tuberculosis, with a Chapter on Gross Post-Mortem and Microscopic Pathology by Philip Hillkowitz, Philadelphia, J. B. Lippincott Company, 1927.

lesion will heal rapidly under antisyphilitic treatment. In a few cases biopsy alone will provide the means of differentiating a syphiloma and a laryngeal neoplasm.

Acromegaly: Acromegaly, as described by Jackson,[11] sometimes produces deformity of the larynx, which may be mistaken for a benign tumor. However, difficulty in the differentiation would be unusual, because of the associated general signs of acromegaly.

Blastomycosis: Blastomycosis of the larynx frequently forms inflammatory infiltrative nodular lesions, which may resemble benign tumors. More frequently the condition is confused with primary laryngeal tuberculosis. The mucous membrane appears whitish, as if it had been touched with silver nitrate. Microscopically multiple abscesses appear; these also simulate tuberculosis. If the microscopic field is shaded, the blastomyces can be seen in the center of the abscess.

Torulosis: Torulosis may produce an ulcerated laryngeal lesion and is usually associated with pulmonary involvement, which simulates a fibroid type of tuberculosis. In cases in which a roentgenogram of the thorax is suggestive of tuberculosis and repeated examinations of the sputum give negative results, this condition should be thought of, although it is extremely rare. Torulas, which are yeastlike parasitic fungi, may appear in the sputum as round fungi or short hairlike rods. These organisms have cultural characteristics that differ from those of the fungi that produce blastomycosis.

TREATMENT

The treatment of benign tumors is based on the size, position and nature of the growth and on the age and general physical condition of the patient.

In adult patients, if the growth is small and accessible, it can be removed with the guidance of the indirect mirror laryngoscope, but in children and in adults of a nervous temperament, suspension laryngoscopy is the most suitable method of visualizing the lesion for treatment. For direct endoscopic laryngeal operations, we almost always use the Lynch suspension apparatus, through which an excellent view of the interior of the larynx can be obtained. It leaves both hands of the operator free to manipulate such instruments as are necessary for removal of the tumor. In cases in which the tumors are large or are situated in the deep tissues of the larynx, thyrotomy or pharyngotomy is necessary to secure adequate exposure. When one of these procedures is undertaken, a preliminary tracheotomy may be performed. This greatly diminishes the postoperative reaction and risk in many cases.

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11. Jackson, C., and Coates, G. M.: The Nose, Throat, and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

At the clinic two methods are generally employed for eradication of these tumors: (1) excision by use of laryngeal forceps or a scalpel and (2) destruction by diathermy (electrocoagulation). In general, radium and roentgen rays have little place in the treatment of benign laryngeal tumors. Diathermy, when used, should not be carried out too vigorously; in the treatment of a benign tumor there is no need of destroying large amounts of normal tissue, which might result in stenosis or at least in impairment of the quality of the voice.

Severe dyspnea should receive prompt attention before any treatment of the tumor is instituted. Usually tracheotomy is required to establish an adequate airway. Patients with marked respiratory embarrassment should not be permitted to continue pulling for air for any length of time, as their general resistance to infection is markedly reduced and pneumonia is likely to ensue.

With few exceptions, all benign tumors of the larynx should be removed, not only because they interfere with phonation and obstruct the larynx but because of the possibility that a few of them may become malignant. Although this possibility is remote, benign tumors of the larynx sometimes do become malignant. On several occasions we have seen small growths in the larynx, which clinically appeared to be the most innocent of benign tumors but which on histologic section were shown to be highly malignant neoplasms. This only reemphasizes the importance of a microscopic examination of every benign tumor of the larynx.

SPECIAL CONSIDERATION OF VARIOUS BENIGN TUMORS OF THE LARYNX

Adenomas.—Adenomas are rare in the larynx. In the older literature they have been mentioned several times, but we have found none described in the more recent articles. Only 1 case of adenoma of the larynx has been observed at the clinic.

CASE 11.—The patient, a man aged 62, had complained of increasing hoarseness for two months before he came to the clinic. The results of laboratory tests were essentially negative. Indirect laryngoscopic examination revealed a firm-appearing pedunculated tumor, 1.5 cm. in diameter, in the subglottic region; the tumor was attached to the lower surface of the anterior two thirds of the right vocal cord. It was associated with a polypoid thickening of the entire left vocal cord (fig. 19). Under anesthesia induced by nitrous oxide and ether, the patient was suspended with the Lynch apparatus; the pedunculated tumor was grasped with forceps and cut off with a laryngeal knife, and the polypoid thickening was removed with biting forceps. Histologic examination of the pedunculated tumor revealed an adenoma that contained a mucous cyst, while examination of the polypoid tumor disclosed myxomatous tissue. The patient made an uneventful recovery.

Papillomas.—Papillomas are the most common of all true benign laryngeal neoplasms, and, since they produce serious symptoms in many instances and have a marked tendency to recur in spite of any type of treatment, it is not surprising that many articles in the literature deal with this important subject (fig. 20).

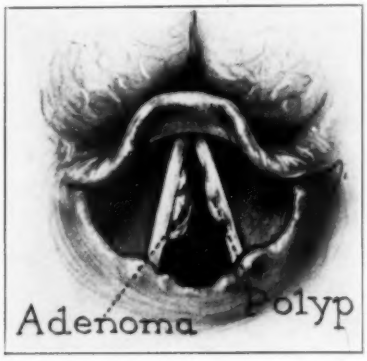

Fig. 19.—Adenoma attached to the anterior two thirds of the right vocal cord and associated with polypoid thickening of the left vocal cord.

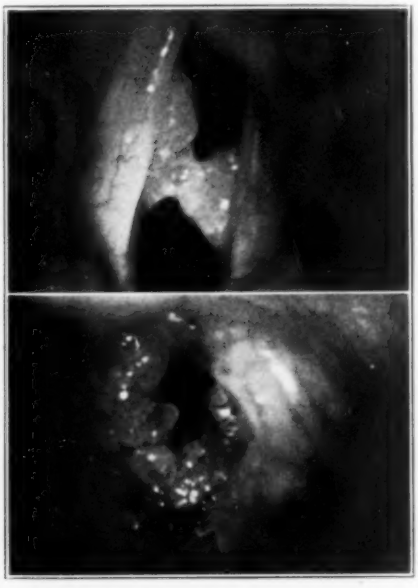

Fig. 20.—Multiple papillomas of the larynx (photographs).

At the clinic 194 cases of papilloma of the larynx have been observed. A good opportunity for study of these cases has been available, as 42 per cent of the patients who stayed for treatment have been under observation for one to five years, 9 per cent for five to ten

years, 7 per cent for ten to nineteen years and 20 per cent for one month to one year. Twenty-one per cent were seen for less than one month.

Papillomas occur more frequently among men than among women. One hundred and twenty-one, or 62 per cent, of our patients were male; 73 or 38 per cent, female. It is frequently stated that these tumors occur more often among children than they do among adults. However, in our experience, the reverse is true; 60 per cent of our patients were 20 or more, while 40 per cent were under 20. Papillomas in children are most frequently encountered between the ages of 1 and 5 years, while in adults they are seen usually between 20 and 35. Our youngest patient was 10 months of age; our oldest patient, 73; 26 patients were between the ages of 45 and 65.

As has been observed by many authors, papillomas which affect children are self limited. We have found that these growths have a tendency to disappear at about the age of puberty. In most of our adult patients, on the other hand, the tumors did not occur until after the age of puberty.

In children papillomas always seem to be multiple, and in adults the same is usually true. We have observed only 28 cases in which a single papilloma was present in the larynx; in each of these cases the patient was an adult.

The most important symptoms of papillomas of the larynx are hoarseness and dyspnea. In none of the 194 cases was hoarseness absent, and in about 45 per cent the patient was troubled with dyspnea. In 35 cases the dyspnea was severe enough to warrant tracheotomy. In 21 other cases tracheotomy had been performed before the patient came to the clinic. Papillomas apparently have much more energy of growth in children than they do in adults. Not only do these tumors involve the larynx more extensively, but they tend to recur more rapidly and more tenaciously in children than in adults. The fact that they involve the larynx more extensively in children is evident from the observation that in 78 per cent of cases in which tracheotomy was performed the patient was a child. Not only do children have recurrences more often than do adults, but the frequency and rapidity of recurrence in a single case are greater in children. This is true in the majority of cases but not in every one. In a few of our adult patients the papillomas recurred frequently after each removal.

Particularly in children there is a marked tendency for papillomas to recur at the site of removal and to develop in other portions of the larynx. Frequently, in spite of treatment, extensions of these papillary tumors are noted on the epiglottis, in the pharynx or in the trachea.

Papillomas can undergo malignant change. This was noted in 3 of our cases in which there had been no previous therapy which might account for the development of malignancy. In all these cases a squamous cell epithelioma, grade 1, was found in situ in a mass of papillomas. Removal of the papillomas cured the 3 patients. It is commonly accepted that implantation of papillomas can and does occur. Hitz and Oesterlin [12] reported a case of multiple papillomas of the larynx in which the patient died of pulmonary metastasis. After a pathologic investigation, it was decided that the condition was due to aerial metastasis.

At the present time no one treatment for papillomas of the larynx is entirely satisfactory. In spite of any form of treatment, these tumors, particularly in children, tend to recur. Many methods, both surgical and medical, have been and are being used in the treatment of this condition, and each method has its staunch supporters. The various forms of treatment may be listed as follows: (1) tracheotomy without any other form of treatment (this method is applicable for children if it can be assumed that the disease is self limited and will disappear whether or not any treatment is instituted); (2) avulsion of the tumors with a forceps, knife, curet, snare or punch; (3) cauterization with the actual cautery; (4) fulguration or electrocoagulation; (5) application of radium; (6) roentgen irradiation; (7) thyrotomy; (8) destruction of the growths with escharotics; (9) medication, and (10) combinations of these methods.

In reference to the treatment of multiple papillomas, a review of the more recent literature is interesting. Jackson [13] expressed the opinion that superficial avulsion of the growths is the only method that will insure a perfect voice and airway after the growths cease to recur. He insisted that radical measures, in children particularly, are unnecessary, since recurrences will appear in spite of any method of treatment. Katz [14] reported good results with the administration of magnesium oxide by mouth in conjunction with endolaryngeal indirect removal of the growths. He claimed that administration of the drug for several months shortens the time of treatment. He condemned the use of roentgen rays, which he said never cured or helped the condition in any case. Hubbard and Galbraith [15] recommended destruction of

12. Hitz, H. B., and Oesterlin, E.: A Case of Multiple Papillomata of the Larynx with Aerial Metastases to Lungs, Am. J. Path. **8**:333-338 (May) 1932.

13. Jackson, C.: Treatment of Benign Tumors and Tumor-Like Conditions of the Larynx, Tr. Am. Laryng. A. **59**:45-51, 1937.

14. Katz, B.: Treatment of Papillomas of the Larynx with Calcined Magnesia, Ann. Otol., Rhin. & Laryng. **41**:202-215 (March) 1932.

15. Hubbard, T., and Galbraith, E. G.: The Treatment of Papilloma of the Larynx by Fulguration and Diathermy, Arch. Otolaryng. **1**:70-78 (Jan.) 1925.

the growths by fulguration and said that transplantation cannot occur if this method is used. Foster [16] expressed the opinion that removal of the growths with a forceps, punch or cautery leads to irreparable injuries of the larynx. He condemned fulguration but said that roentgen rays in proper doses cause prompt disappearance of the growths and provoke no disagreeable reaction. Clerf [17] condemned radiation, as it leads to perichondritis, cicatricial stenosis or atresia. Romonek [18] said that the most successful treatment in his hands is surgical removal of the growths, followed by radium therapy.

New and Kirch [19] have reviewed the foreign literature on this subject. Munyo [20] stated that radium was more dangerous than beneficial. He concluded that roentgen therapy is the only treatment that will cure this disease. Gruschetzkaja [21] reported 7 cases of papillomas in which good results were obtained with the use of roentgen rays. Spira [22] reported 6 cases and said that roentgen therapy is the treatment of choice. Tempea and Gilbuzoianu [23] favored high voltage roentgen therapy, which they said acts rapidly and permanently. They said that radiation must be given in sufficient dosage, or it will act as an irritant. Dahmann [24] expressed the opinion that surgical intervention is the most favorable treatment and may be followed by the application of nitric acid. He said that when diathermy is used it should be of the unipolar type, which limits the depths of coagulation. He opposed irradiation and open operations. Runge [25] advocated the use of roentgen rays in small doses for children but advised surgical procedures for adults followed by the use of nitric acid or roentgen rays. Thrane [26] favored roentgen therapy. He said that it is necessary

16. Foster, J. H.: Papillomatosis Laryngis, Ann. Otol., Rhin. & Laryng. **42**:548-559 (June) 1933.

17. Clerf, L. H.: Laryngeal Complications of Irradiation, Arch. Otolaryng. **6**:338-345 (Oct.) 1927.

18. Romonek, P. L.: Laryngeal Papillomatosis, Nebraska M. J. **19**:130-134 (April) 1934.

19. New, G. B., and Kirch, W.: (*a*) Tumors of the Nose and Throat: A Review of the Literature, Arch. Otolaryng. **8**:600-607 (Nov.) 1928; (*b*) Tumors of the Nose and Throat, ibid. **9**:445-450 (April) 1929; (*c*) Tumors of the Nose and Throat: A Review of the Literature, ibid. **11**:657-668 (May) 1930; (*d*) Tumors of the Nose and Throat, ibid. **17**:563-582 (April) 1933; (*e*) ibid. **19**: 621-637 (May) 1934; (*f*) ibid. **26**:96-109 (July) 1937.

20. Munyo, C., cited by New and Kirch.[19a]

21. Gruschetzkaja, M., cited by New and Kirch.[19a]

22. Spira, cited by New and Kirch.[19b]

23. Tempea, V., and Gilbuzoianu, cited by New and Kirch.[19c]

24. Dahmann, H., cited by New and Kirch.[19c]

25. Runge, H., cited by New and Kirch.[19c]

26. Thrane, K., cited by New and Kirch.[19c]

to use small doses and to be patient, as the effect will last for many months. Schmiegelow [27] said that any form of treatment of papillomas in children is unsatisfactory and may be dangerous. He advised tracheotomy when necessary and expressed the opinion that the condition will clear up spontaneously. Pfeiffer [28] warned against the use of fuming nitric acid, advocated by other German investigators. Koch [29] found that the best results were obtained by the use of roentgen rays after surgical treatment. He said that papillomas differ in sensitivity to roentgen rays and that the treatment should be varied accordingly. Those made up of closely knit cellular stroma respond best to irradiation.

These reports of the superiority and inferiority of the various methods of treatment are conflicting, but observation and treatment of papillomas of the larynx during a long period by one of us (G.B.N.) has led us to adopt one form of treatment in preference to other methods that have been tried at the clinic.

We have had no experience with internal medication as a therapeutic measure, and we do not favor the use of escharotics or of the actual cautery. Roentgen rays have proved unsatisfactory in our hands, although there is some evidence of late to indicate that roentgen therapy in proper dosage is of decided value in the treatment of papillomas. For several years radium was used with excellent immediate results, and in some cases the papillomas disappeared permanently. However, the subsequent scarring that occurs in the larynx, in some cases years later, contraindicates the use of radium. Moreover, several patients who had been treated elsewhere by irradiation were found to have cicatricial stenosis of the larynx when they were examined at the clinic. Consequently, irradiation has been entirely discontinued. From a review of the literature, it would seem that those who advocate the use of radium have not observed their cases a sufficient length of time to discover its disadvantages.

For the past few years diathermy (electrocoagulation) has been used almost entirely at the clinic in the treatment of papillomas. By using a small amount of current, which is carefully controlled, each individual papilloma can be lightly touched with the positive electrode; this method quickly destroys the tumor without affecting the underlying tissues. It has the advantage of being a bloodless procedure, which allows clearer visibility of the field of operation. Transplantations are not as likely to follow electrocoagulation as avulsion. We have observed no cases in which scarring in the larynx followed diathermy or in which the voice was disturbed.

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27. Schmiegelow, E., cited by New and Kirch.[19d]
28. Pfeiffer, W., cited by New and Kirch. [19e]
29. Koch, J., cited by New and Kirch.[19f]

In cases of multiple papillomas in which there is no evidence of laryngeal obstruction, we have adopted the method of treating one side of the larynx at a time. During the first operation all the papillomas on one side are removed by diathermy, and not until this side has entirely healed are the papillomas on the opposite side removed.

In children we prefer to treat the papillomas as they develop; in several cases, after two or three treatments with diathermy the tumors did not recur. Every thyrotomy involves a certain amount of risk, and we feel that this operation is usually unnecessary in the treatment of papillomas unless malignancy is suspected.

Fibromas.—Only 7 cases of true fibroma of the larynx have been observed at the clinic. In each of these cases the patient was a man; the ages ranged from 25 to 52. In only 1 case, which will be discussed subsequently, did the patient complain of any symptom other than hoarseness. In these cases the hoarseness had been present for nine months to seventeen years.

Clinically, these tumors possess no features that distinguish them from many other benign growths of the larynx. In 3 cases the growths were polypoid; in 2 cases the growths were vascular, and in the remaining cases, in which the growths were large, they appeared firm and fibrous. The tumors varied in size from 2.5 by 2.5 mm. to 2.5 by 4 cm. Three were situated on the anterior third of the right vocal cord; 1 was situated at the junction of the anterior and the middle third of the left vocal cord; 1 was connected to the anterior commissure in the subglottic region (fig. 21*A*), and 1 was attached to the posterior surface of the epiglottis. The point of attachment of the remaining growth was not stated in this history. In the treatment of these tumors, 4 were removed with the guidance of the indirect laryngoscope, and 3 were removed under suspension laryngoscopy. To our knowledge, a recurrence of the growth did not occur in any of these cases.

In the following case the tumor was of special interest because of its size and situation. This case was reported by New in 1929.[30]

Case 12.—The patient, a man of 25, complained not of hoarseness but of an increasing difficulty in speaking clearly; this difficulty had been noted first eight years before the patient came to the clinic. At times he had had some difficulty in swallowing and had been aware of the sensation of a lump in his throat. After an examination of his larynx, it was surprising to learn that he had had remarkably little dyspnea. On indirect laryngoscopic examination a large, firm tumor, 2.5 by 4 cm., was visible; it was attached to the posterior surface of the epiglottis and almost completely filled the hypopharynx (fig. 21 *B*). No ulceration of the mucous membrane covering the tumor was present. Owing to the size of the lesion, a preliminary tracheotomy was performed, and twelve days later

30. New, G. B.: Fibroma of the Larynx, S. Clin. North America **9**:82-84 (Feb.) 1929.

the greater part of the tumor was destroyed by surgical diathermy under suspension laryngoscopy. About six weeks later the remaining portion of the growth was similarly destroyed. The tumor never recurred, and when the patient was last seen there was no evidence of the growth, and the larynx appeared normal except for slight thickening of the epiglottis.

This was the largest tumor of its type that we have ever seen. Chappell [31] in 1907 reported a case in which the condition was similar to that just described. In his case the tumor was removed by subhyoid pharyngotomy. We believe that destruction of such a neoplasm by surgical diathermy under suspension laryngoscopy is the method of choice, as compared to removal of the growth by pharyngotomy, because with

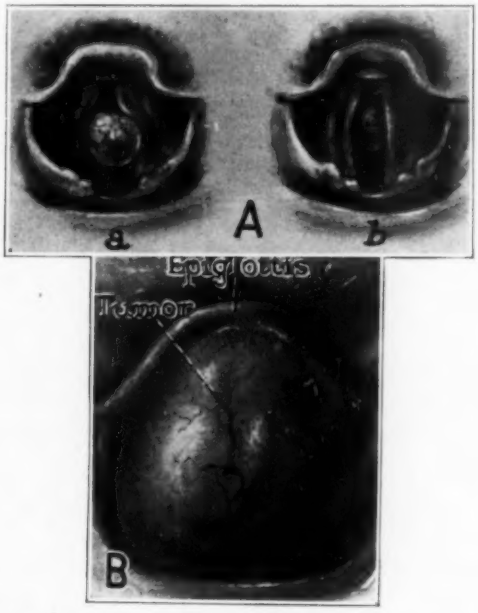

Fig. 21.—*A*, pedunculated fibroma attached to the anterior commissure in the subglottic region; *a*, the tumor is forced upward between the cords on expiration; *b*, the tumor lies below the cords during inspiration. *B*, fibroma of the epiglottis.

the former procedure the operative risk is reduced to a minimum; the postoperative reaction is less severe; the period of convalescence is shortened, and no external or internal scarring results.

Neurofibromas.—Neurofibroma of the larynx is exceedingly rare. We have observed but 1 case.

Case 13.—The patient was a woman aged 49. For twelve years she had had intermittent attacks of hoarseness, which gradually had increased in severity.

31. Chappell, W. F.: Large Tumor of the Laryngo-Pharynx Removed by Sub-Hyoid Pharyngotomy, Tr. Am. Laryng. A. **29**:98-104, 1907.

She had not had any other symptom. The results of routine laboratory tests and a general physical examination were essentially negative. Examination of the larynx with the indirect mirror laryngoscope revealed a fibrous tumor which measured 3 by 8 mm. and was attached to the free border of the middle third of the right vocal cord (fig. 22). The patient was suspended under anesthesia induced by nitrogen monoxide and ether. The growth was removed with laryngeal forceps, and the base was electrocoagulated. Microscopic examination disclosed that the lesion was a neurofibroma.

This lesion apparently was solitary and was not associated with other signs of neurofibromatosis. Jackson and Coates [11] reported a case of similar involvement in a woman of 23, who also did not have any similar lesions elsewhere in the body.

Lipomas and Fibrolipomas.—Lipomas are seen but rarely in the larynx. Only 1 such tumor has been observed at the clinic. Chevalier Jackson has seen 7 laryngeal lipomas in forty years of practice.

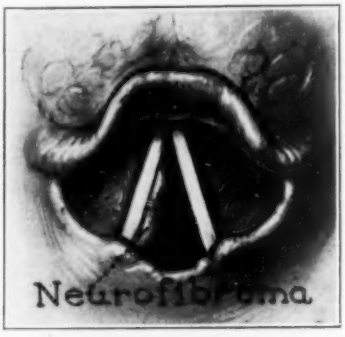

Fig. 22.—Neurofibroma attached to the anterior and the middle third of the right vocal cord.

There is much controversy as to the causation of lipomas and fibrolipomas. In a comprehensive account Goldstein [32] described three theories concerning the cause of these neoplasms. Briefly, these theories may be stated as follows: 1. A lipoma may be formed by encapsulated fat cells or small patches of adipose tissue, which have undergone simple hyperplastic changes. 2. A lipoma may have an embryonic origin, in which an invagination of mesodermal tissue develops into a tumor composed essentially of fat. 3. A lipoma may be produced by a disturbance in the chemistry of normal cells, in which fatty acids, instead of being excreted as a by-product, are retained in the cells as fat. Goldstein mentioned the possibility that lipomas actually may be fibromas which have undergone fatty changes as a result of physiochemical activity.

32. Goldstein, M. A.: Lipoma of the Larynx, Laryngoscope **19**:641-653 (Sept.) 1909.

Dixon and Helwig [33] favored the last of these theories and expressed the opinion that the lipoma which they reported was due to an infiltration of fat into the submucous laryngeal stroma following a chronic inflammatory reaction. This inflammatory process initiates chemical activity that results in the deposition of fat in the involved region. Our patient, who had a fibrolipoma, stated that he had had several attacks of laryngitis; these may have produced in the larynx a chronic inflammatory process, which, in turn, may have paved the way for the development of the lipoma. However, this is entirely a matter of conjecture.

A review of the literature indicates that laryngeal lipomas may occur at any age and that more of these tumors are extrinsic than intrinsic in relation to the larynx. Birkett [34] quoted Jackson as stating that in his 7 cases only 1 tumor was intrinsic.

Lipomas may be pedunculated in the larynx, and when of sufficient size they will induce marked dyspnea. Goldstein reported such a tumor, a large round growth with an attachment in the left ventricular fossa. Imperatori [35] reported a pedunculated lipoma, 7 by 6.6 by 4.7 cm., which was attached by a narrow base to the right arytenoepiglottic fold; this lesion filled much of the hypopharynx and almost occluded the vestibule of the larynx.

Frequently a lipoma may be situated beneath the mucosa and produce expansion of some anatomic portion of the larynx. In our case the tumor distended the left arytenoepiglottic fold. Davis [36] reported a lipoma which caused a globular swelling of the right arytenoepiglottic fold and the false vocal cord; this growth measured 1⅛ by ⅝ inches (2.9 by 1.6 cm.). Birkett reported a case in which the tumor, which measured 4.5 by 4.5 by 2.5 cm., expanded the false vocal cord on the left side.

When of large dimensions, a lipoma lying between the submucosa of the larynx and the thyroid cartilage may push the involved side of the larynx inward, produce much distortion, diminish the airway and cause marked dyspnea. Flynn [37] observed a case of this type in which the lipoma pushed the base of the tongue and the entire epiglottis backward so that the vestibule of the larynx could not be visualized

33. Dixon, O. J., and Helwig, F. C.: Lipoma of the Larynx, Arch. Otolaryng. **14**:284-290 (Sept.) 1931.

34. Birkett, H. S.: Lipoma of the Larynx Intrinsic in Orgin, J. Laryng. & Otol. **49**:733-740 (Nov.) 1934.

35. Imperatori, C. J.: Fibrolipoma of the Larynx: Report of a Case, Laryngoscope **43**:940-944 (Nov.) 1933.

36. Davis, E. D. D.: A Lipoma of the Larynx, J. Laryng. & Otol. **48**:824-825 (Dec.) 1933.

37. Flynn, J. A.: Extrinsic Lipoma of the Larynx, Laryngoscope **43**:294-297 (April) 1933.

during laryngoscopic examination. In a case reported by Walford,[38] the tumor measured 3½ by 4 inches (8.9 by 10.2 cm.) and was dumbbell shaped as a result of a constriction caused by the right superior horn of the thyroid cartilage and the hyoid bone; the larger portion of this mass was situated in the neck, external to the larynx, while the smaller part lay on the inner aspect of the right ala of the thyroid cartilage and pushed the mucous membrane of the larynx and pharynx inward.

The symptoms caused by the presence of lipoma in the larynx are not characteristic. In some instances one may be able to diagnose a pedunculated lipoma clinically, but with the extrinsic types one can never be certain of the nature of the lesion until it is removed or until a specimen is removed for biopsy.

The treatment of laryngeal lipoma must necessarily depend on the size and situation of the tumor. If the growth is pedunculated, it may be removed with laryngeal forceps or a snare under suspension laryngoscopy. If the lipoma is extrinsic and produces displacement of the internal structures of the larynx, we feel that it is preferable to explore the growth through an external incision, as the diagnosis of such a tumor can never be established with certainty preoperatively. If adequate exposure is secured, any form of treatment then can be undertaken to eradicate the tumor, regardless of its nature. In cases in which dyspnea is a prominent symptom owing to laryngeal obstruction, it is well to perform a preliminary tracheotomy before instituting any form of treatment directed toward removal of the growth.

CASE 14.—The patient, a woman aged 29, had had intermittent attacks of hoarseness, presumably caused by laryngitis, for eight years, and three months previous to her arrival at the clinic she had had spasms of the throat and some difficulty in breathing. When she came to the clinic she had slight dyspnea. The results of laboratory tests and a general physical examination were negative. Laryngeal examination disclosed a tumor 1 inch (2.5 cm.) in diameter, which was covered with normal mucous membrane and bulged the left arytenoepiglottic fold (fig. 23). It was elastic and could be indented with a probe. Owing to the size and position of the growth, thyrotomy was performed, and the tumor was easily shelled out. It measured 2.5 by 2.5 cm., and microscopic examination revealed that it was fibrolipoma. The patient made a complete recovery, except for slight residual scarring, which resulted in some limitation of movement of the left vocal cord.

Chondromas and Osteochondromas.—Chondromas of the larynx are of interest because of their character and rarity. Eight such tumors have been seen at the clinic; 1 of these was reported by New [2] and 6 by Figi.[39]

38. Walford, A.: Lipoma of the Larynx, J. Laryng. & Otol. **45**:548-550 (Aug.) 1930.

39. Figi, F. A.: Chondroma of the Larynx: Report of Six Cases, Ann. Otol., Rhin. & Laryng. **41**:369-386 (June) 1932.

All cases of chondroma of the larynx which have been reported were reviewed by New [2] in 1918, by Moore [40] in 1925 and again by Figi in 1932.[39]

In the literature much attention has been given to the pathologic structure and the classification of these cartilaginous tumors; the classification has been rather briefly described in this paper. All the 8 chondromas which were observed at the clinic were considered true tumors. Two of these neoplasms were found in women and the remaining 6 in men. The ages of the patients varied from 34 to 65, and the average age was 52.

The great majority of chondromas of the larynx involve the thyroid or the cricoid cartilage and usually arise from its inner surface. A few have been recorded as originating from the epiglottis or from the arytenoid cartilages. In 4 of the 8 cases the tumor was attached to the thyroid cartilage and in 4 to the cricoid cartilage.

Fig. 23.—Fibrolipoma bulging the left arytenoepiglottic fold.

Since laryngeal chondromas grow slowly, the symptoms which they produce develop insidiously and gradually increase in severity. The duration of symptoms in the 8 cases varied from seven months to five years. Although extremely variable, the most prominent symptoms are concerned with respiration and deglutition. In all our cases the patient complained of shortness of breath, and in 3 cases dyspnea was the initial symptom. The degree of dyspnea depends on the size and position of the tumor; the inward growing chondromas arising from the cricoid cartilage are likely to obstruct the respiratory tract to a greater extent than those which are attached to the thyroid cartilage. A chondroma arising from an arytenoid cartilage may produce little or no

40. Moore, I.: Cartilaginous Tumors of the Larynx: A Study of All the Recorded Cases, J. Laryng. & Otol. **40**:9-14 (Jan.); 84-109 (Feb.); 145-164 (March) 1925.

dyspnea but may interfere with swallowing. On laryngeal examination in a case in which but little dyspnea is complained of it is often surprising to find a large obstructing chondroma, which has reduced the laryngeal lumen to a mere slit. By way of explanation, the diminished amount of air causes the patient only a small amount of discomfort because the tumor and associated obstruction develop so slowly that the patient gradually becomes accustomed to and learns to tolerate it. However, with more rapidly growing chondromas, dyspnea may be a serious symptom; several cases have been reported in the literature in which the patient died of dyspnea, and many patients with laryngeal chondromas have required emergency tracheotomy. Marked narrowing of the laryngeal lumen may result in stridor, which is particularly noticeable no inspiration.

When the tumor lies in the hypopharynx or is situated adjacent to the esophagus, mechanical interference with deglutition results. One of our patients experienced a choking sensation on swallowing, especially when fluids were ingested.

Hoarseness is a common but not an outstanding symptom. All but 1 of our patients complained of hoarseness, which is due to mechanical interference with the normal movements of one or both vocal cords. Such interference may amount to nothing more than slight impairment of motion or may cause actual fixation of a vocal cord. In only 3 of our cases was a fixed vocal cord discovered. (In 1 case paralysis of both vocal cords was noted.) As in the case of dyspnea, the degree of hoarseness depends largely on the size and situation of the growth. Chondromas attached to the cricoid, the arytenoid or the epiglottic cartilage are likely to cause but little hoarseness, while those originating from the thyroid cartilage grow inward to involve the adjoining vocal cord, which eventually becomes fixed.

When a chondroma of the larynx is of large dimensions, a sense of fulness or pressure in the neck may be experienced by the patient. Only 1 of our patients complained of cough.

Chondromas which grow from the external aspect of the thyroid or the cricoid cartilage give rise to a palpable or even visible tumor in the neck; this occurred in 3 of our cases. In each of these cases, the chondroma was attached to the right ala of the thyroid cartilage.

Since chondromas of the larynx are encountered infrequently, the diagnosis may offer some difficulty. An examination of the neck may reveal fulness of the thyroid or the cricoid cartilage or an actual mass. On palpation such a tumor has a nodular surface and is extremely hard. Strongly suggestive of a chondroma is an external tumor that is fixed to one of the laryngeal cartilages, is not tender, is hard on palpation and is unassociated with signs of inflammation or adenitis. On laryngoscopic examination, if the growth extends into the laryngeal lumen, a

rounded tumor is seen; this is covered with normal unulcerated mucous membrane in which blood vessels stand out prominently. Impaired mobility or actual fixation of one or both vocal cords may be noticeable. A probe passed into the larynx will give evidence of the hard character of the mass. Difficulty in diagnosis may be encountered if the lesion is large and arises from one lamina of the thyroid cartilage and pushes all the internal structures of the larynx to the opposite side. Furthermore, a chondroma in the subglottic region may be entirely overlooked.

Of particular importance as diagnostic evidence of a chondroma is the use of the roentgenogram. If of sufficient density, the tumor will stand out as an area of increased density on the roentgenogram; even if the growth is not calcified, its outline may be visible on properly exposed roentgenograms. As expressed by Figi,[39]

> Unless the roentgenograms are taken with the possibility of a chondroma of the larynx in mind, however, the tumor may not be recognized, due to overexposure of the film. This is more likely to occur if, as is frequently the case, there is no calcification of the growth.

In 5 of our 8 cases roentgenograms were made, and the outline of the tumor was visible in each roentgenogram.

The history and the physical, laryngeal and roentgenologic findings should offer sufficient evidence to justify the diagnosis of chondroma. However, one should always substantiate these clinical findings with a microscopic examination of the tumor.

The method of treatment of a chondroma of the larynx depends on the size and situation of the lesion. Some laryngologists have removed small pedunculated chondromas and those attached to the epiglottis with the aid of the indirect or direct laryngoscope or under suspension laryngoscopy. However, in none of our cases were such methods considered feasible. In 4 of our cases the growth was situated on the inner aspect of the thyroid or the cricoid cartilage, and these chondromas were removed satisfactorily by thyrotomy. In 2 cases the growth was situated externally and involved the thyroid cartilage; in operation through an external incision it was found necessary to remove almost the entire right lamina of the thyroid cartilage. In both cases a thin shell of cartilage was left in place to maintain the patency of the laryngeal lumen. In 1 case, owing to the extensiveness of the growth, it was decided to apply radium and observe the patient for a few weeks. However, the patient did not return for further treatment. Radium seems to be of little value in the treatment of these cartilaginous tumors. Laryngectomy should be performed only when the growth is so extensive that to effect a cure would necessitate the removal of all or almost all the cartilaginous support of the larynx. In 1 of our cases laryngectomy was required.

Recurrences of the growth after removal are unlikely if the chondroma has been thoroughly eradicated. In general, a chondroma should be entirely removed, even if radical measures are required. Conservative methods of treatment are sometimes feasible for extensive growths, when radical removal of the tumor would result in a loss of most of the cartilaginous support of the larynx and, in turn, allow for a laryngeal collapse. In such cases the main bulk of the tumor may be removed with the anticipation that it will recur slowly, thus giving the patient the use of his larynx for several years.

In the drawings (figs. 24, 26 and 28) we have attempted to show somewhat diagrammatically each of our 8 chondromas in relation to the thyroid and the cricoid cartilage and to the vocal ligaments. A short résumé of each case has been added.

CASE 15.—A woman aged 64 had had dyspnea for four or five years. About ten months before she came to the clinic she had noticed a tumor in the right side of her neck. Examination of the larynx disclosed a large hard nodular tumor, which was attached to the right side of the thyroid cartilage (fig. 24 *A*). The tumor had caused almost complete obstruction of the larynx. Laryngectomy was performed because of the extent of the lesion. The patient died of pneumonia five days after the operation. Microscopic examination revealed that the tumor was a chondroma.

CASE 16.—The patient was a man aged 44. A tumor had developed on the right side of his neck four years before he came to the clinic. The size of the tumor had increased progressively. He had had dyspnea for one year. The severity of the dyspnea had increased, and tracheotomy had been performed six months before he came to the clinic. Laryngeal examination disclosed a large hard nodular tumor, which was attached to the right ala of the thyroid cartilage (fig. 24 *A*). The tumor had bulged into the larynx above the vocal cords. A tracheotomy tube was in place. The tumor was enucleated through an external incision in the neck. A shell of the mesial side of the tumor was left to give support to the soft tissues on the right side of the larynx. The tumor involved the full thickness of the right lamina of the thyroid cartilage. Microscopic examination revealed that the tumor was a chondroma. When the patient was examined two years after operation, there was no evidence of recurrence of the tumor.

CASE 17.—A man aged 57 had noticed a lump in the right side of his neck three years before he came to the clinic. The size of the lump had increased gradually. Hoarseness and dyspnea had been present for six months; the severity of these symptoms had increased. Laryngeal examination disclosed a large hard nodular tumor, which was attached to the right ala of the thyroid cartilage (fig. 24 *A*). This mass had displaced the tissues on the right side of the larynx markedly to the left. The right vocal cord was fixed. The treatment was the same as that employed in case 16. Microscopic examination revealed that the tumor was a chondroma. When the patient was examined fifteen months after the operation there was evidence of recurrence of the tumor. Laryngectomy was advised because of the extent of the recurrent tumor, but the patient refused to submit to this operation. We have not seen him since.

CASE 18.—A man aged 34 had noticed hoarseness and slight dyspnea for ten months before he came to the clinic. He also had had some difficulty in

swallowing liquids. Laryngeal examination disclosed a smooth round tumor in the subglottic region, situated to the left of and posterior to the cricoid cartilage (fig. 24 *B*). There was no ulceration. Both vocal cords moved normally. Thyrotomy was performed, and the tumor was enucleated. Microscopic examination revealed that the tumor was an osteochondroma. There was no evidence of recurrence when the patient was seen three years after the operation.

CASE 19.—A woman aged 57 had complained of increasing hoarseness and dyspnea for ten months before she came to the clinic. She had had choking sensations when she swallowed, particularly when she swallowed liquids. Laryngeal examination disclosed a rounded tumor, which was situated posteriorly in the right subglottic region (fig. 24 *C*). There was no evidence of ulceration.

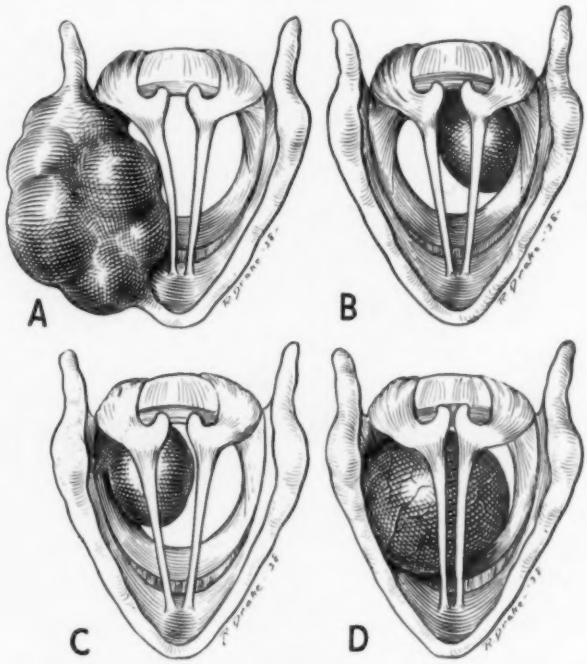

Fig. 24.—*A*, chondroma involving the substance of the right ala of the thyroid cartilage. *B*, chondroma arising from the cricoid cartilage. *C*, chondroma arising from the cricoid cartilage. *D*, chondroma arising from the lower half of the right ala of the thyroid cartilage; tumor situated below the vocal ligaments.

Roentgen examination confirmed the presence of the tumor. Radium therapy was employed, in order that the tumor might be watched for a few weeks. The patient never returned to the clinic. She died three and a half years later; her death presumably was due to the tumor.

CASE 20.—A man aged 42 had had intermittent hoarseness for three and a half years and persistent hoarseness and dyspnea for eight months before he came to the clinic. He also had had severe cough. Laryngeal examination disclosed bilateral fixation of the vocal cords. Tracheotomy was performed. The patient had syphilis, which accounted for the paralysis of the vocal cords. Anti-

syphilitic therapy was employed. A roentgenogram of the larynx disclosed what appeared to be a chondroma, situated in the subglottic region (figs. 24 *D* and 25). Thyrotomy was performed, and a spherical tumor, 2.5 cm. in diameter, was removed. The tumor was attached to the lower half of the thyroid cartilage on the right side. The free margin of the tumor was in contact with the laryngeal wall on the opposite side. Microscopic examination revealed that the tumor was a chondroma. When the patient was seen ten months later the right vocal cord moved normally, but a tracheal tube had to be worn because of persistent paralysis of the right vocal cord. There was no evidence of recurrence of the tumor.

Case 21.—A man aged 58 had had a husky voice for six months and slight dyspnea for a few days before he came to the clinic. Laryngeal examination disclosed a smooth rounded sessile tumor which measured 1 by 2 cm., situated in

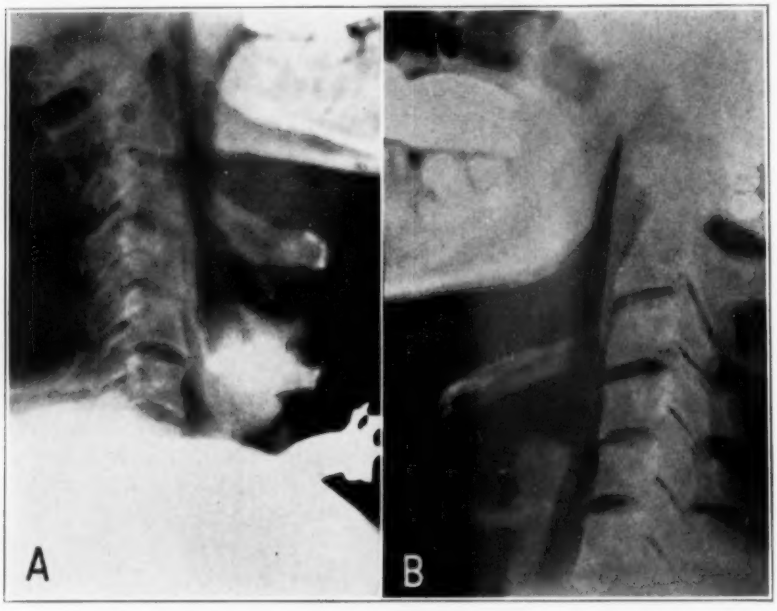

Fig. 25.—*A*, roentgenogram showing a subglottic chondroma. *B*, roentgenogram taken after removal of the chondroma shown in *A*.

the interarytenoid portion of the subglottic region and attached to the cricoid cartilage (figs. 26 and 27). Thyrotomy was performed, and the tumor was enucleated. Microscopic examination revealed that the tumor was a chondroma. There was no evidence of recurrence of the tumor when the patient was seen sixteen months later.

Case 22.—A man aged 65 had had increasing dyspnea for seven months before he came to the clinic. Laryngeal examination disclosed a tumor which was attached posteriorly to the cricoid cartilage (fig. 28). The tumor, which was 2.5 cm. in diameter, filled the subglottic region. Preliminary tracheotomy was performed. Subsequently, thyrotomy was performed, and the tumor was enucleated. Microscopic examination revealed that the tumor was a chondroma which contained foreign body giant cells. The tumor has not recurred.

Angiomas.—A study of angiomatous tumors of the larynx deserves much consideration, since mismanagement in the treatment of some of these growths can provoke symptoms of a serious nature. This discussion is confined to true simple and cavernous hemangiomas and lymphangiomas and is not concerned with vascular inflammatory tumors or other types of pseudoangiomas.

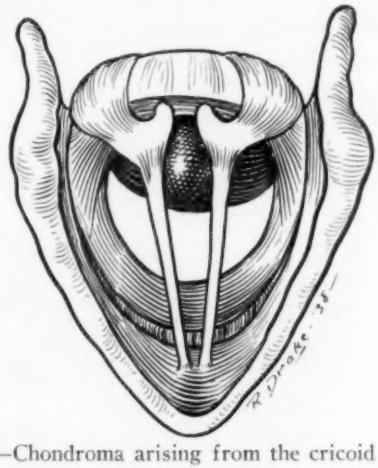

Fig. 26.—Chondroma arising from the cricoid cartilage.

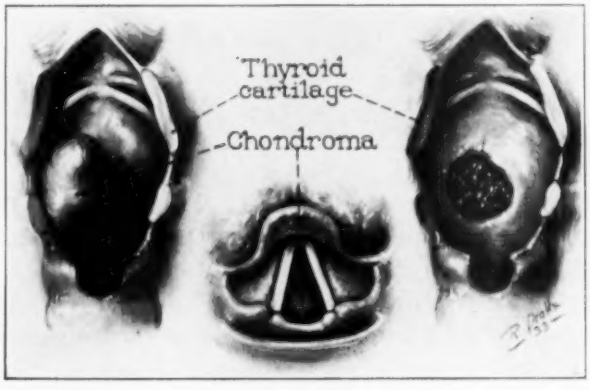

Fig. 27.—Interior of a larynx exposed by thyrotomy before and after the removal of a chondroma.

Howarth [41] expressed the opinion that hemangiomas of the larynx are not rare and said that many cases observed by laryngologists are not reported. However, only 25 cases of true angioma of the larynx

41. Howarth, W.: A Note on Hemangeiomata of the Larynx, J. Laryng. & Otol. **45**:803-804 (Nov.) 1930.

have been observed at the clinic. In 1 case the tumor was a lymphangioma; in 3 cases, a cavernous hemangioma, and in the remaining 21 cases, a simple hemangioma. The ages of the patients varied from 11 weeks to 57 years. Aside from 2 infants, all the patients were adults; 64 per cent of the patients were between the ages of 25 and 45; 68 per cent were male and 32 per cent female.

The symptoms produced by angiomas of the larynx do not differ essentially from those caused by other types of benign tumor. Hoarseness is a common symptom. Intermittent or persistent hoarseness was present in all but 1 of the cases in which the patient was adult. Whether or not dyspnea is present depends on the type, size and situation of the tumor. Angiomas in infants and children are likely

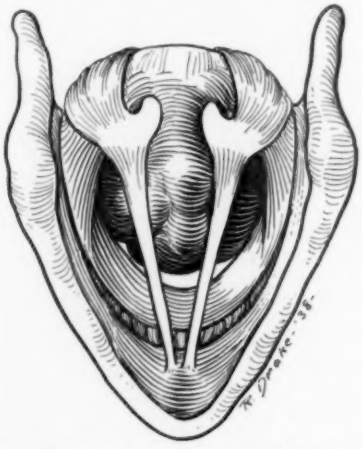

Fig. 28.—Chondroma arising from the cricoid cartilage.

to be cavernous, large, sessile and productive of marked respiratory difficulty. Both infants had dyspnea; in 1 the obstruction of the larynx caused slight cyanosis. The majority of angiomas in adults are small pedunculated growths, which cause little or no interference with respiration. Only 2 of our adult patients complained of dyspnea; in 1 of these the cause was entirely functional. When the tumor involves the epiglottis or arytenoid cartilages, the patient may experience some degree of dysphagia.

In every case of laryngeal angioma there is danger of hemorrhage, which may prove fatal in some instances. However, hemoptysis occurred in only 2 of our cases. In neither of these cases was the hemorrhage severe, although frequent attacks of bleeding occurred in 1 case. Aside from hemoptysis, symptoms caused by angiomas are of no diagnostic significance, and the diagnosis must be based on the clinical appearance of the tumor.

Sweetser [42] has divided angiomas into two types: (1) the adult type, usually rounded, projecting, sometimes pedunculated and situated on or above the vocal cords (fig. 29); (2) the infantile type, sessile, flattened and diffuse, occurring below the vocal cords and generally producing dyspnea. The 25 laryngeal angiomas seen at the clinic conform well to Sweetser's classification. The 2 that occurred in infants were sessile and subglottic and produced dyspnea; of the 23 which affected adults, all but 1, which was a rounded subglottic nodule, were projecting and occurred on or above the vocal cords. Of those occurring in adults only 1 produced noticeable dyspnea, an angioma so large as to fill the supraglottic space almost completely.

Cavernous hemangiomas, which are the most common type of laryngeal angiomas that affect infants, are dark bluish or purplish and rarely

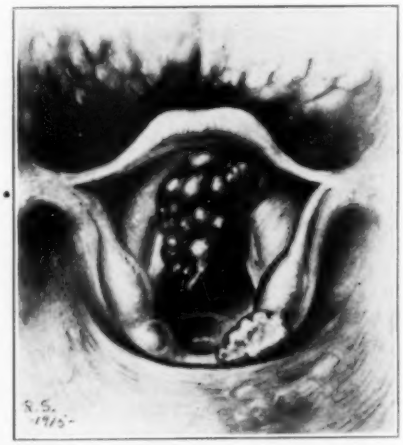

Fig. 29.—Hemangioma of the vocal cords.

pulsate. Although some cavernous hemangiomas have been reported as pedunculated tumors which possess a broad base, they usually are flattened sessile lesions. They have a smooth surface and undergo flattening or a reduction in size when pressure is made against them with a probe. Frequently these lesions are of sufficient size to produce respiratory obstruction.

Cavernous hemangiomas in the larynx of infants often are associated with multiple cavernous hemangiomas elsewhere in the body. One of our patients, an infant aged 11 weeks, who had a subglottic cavernous hemangioma, also had a similar lesion on the right side of the nose, one in the right inner canthus, one over the left breast and one on the scalp. In some infants and children it is not possible to visualize

42. Sweetser, T. H.: Hemangioma of the Larynx, Laryngoscope **31**:797-806 (Oct.) 1921.

a tumor in the subglottic region by any laryngoscopic method and the presence of dermal angiomatous lesions is a decided aid in making the diagnosis of an associated laryngeal angioma.

We have observed 3 cases of cavernous angioma of the larynx; in 2 cases the patient was an infant, and in 1 case the tumor was situated in the left arytenoid region of a woman of 45.

Simple hemangiomas are usually not flattened lesions, as are the cavernous tumors, but are projecting. Ten of the 22 simple angiomas of the larynx seen at the clinic were nodular tumors; 6 were polypoid tumors; 5 were definitely pedunculated, and 1 was a papillary growth involving the entire left vocal cord. These tumors may have a bluish, reddish or pale pink color. In a few cases the color is not suggestive of hemangioma, which makes the diagnosis difficult, if not impossible. The simple hemangiomas that we have seen varied from 3 mm. to 2.5 cm. in diameter, most of them being 5 to 6 mm. in diameter. All but 2 of these tumors were located on the anterior two thirds of the vocal cords; 1 was attached in the subglottic region below the right vocal cord, and the attachment of the other tumor, which almost completely filled the supraglottic space, could not be determined.

The majority of lymphangiomas of the larynx that have been reported in the literature have had a smooth or papillary surface. They are relatively larger than hemangiomas, are pale and transparent and on examination with a probe offer more resistance than do hemangiomas. However, the 1 lymphangioma seen at the clinic was a small polypoid nodule situated at the junction of the anterior and the middle third of the right vocal cord. Lymphangiomas are rare laryngeal tumors.

Ulceration of the mucous membrane covering an angioma is unusual. In 2 of our cases ulceration was noted. In 1 of these cases the ulceration was indistinguishable from malignant ulceration.

Generally it is impossible to distinguish by clinical methods a vascular inflammatory tumor from an angioma. In such cases the tumor should be treated as an angioma. A varix usually is bright red and may pulsate.

Hemorrhage is the most serious complication of angiomas. Regardless of what therapeutic measure is employed, it must meet two qualifications: It must eradicate the growth, and it must prevent or control bleeding.

Some of the smaller projecting simple hemangiomas may be removed safely with laryngeal forceps, but simple hemangiomas of large dimensions are best destroyed by diathermy, which effectively and simply controls any hemorrhagic tendency. However, we agree with Sweetser [42] and Simpson,[43] who favored the use of radium for large simple

43. Simpson, F. E.: Radium in the Treatment of Hemangioma of the Larynx, J. A. M. A. **96**:342-344 (Jan. 31) 1931.

hemangiomas and for cavernous hemangiomas. In the 2 cases of cavernous laryngeal hemangioma in an infant radium was used therapeutically with excellent results. In the case in which a large simple hemangioma almost filled the supraglottic space, radium needles were inserted directly into the tumor; a total dose of 300 mg. hours was given; this caused a 90 per cent reduction in the size of the tumor within two months.

Like other laryngeal neoplasms, angiomas occasionally produce sufficient respiratory obstruction to necessitate tracheotomy for the relief of dyspnea. When tracheotomy is required it should be performed at the lowest possible site, particularly if the lesion is subglottic. This procedure is preferable to intubation, because of the danger of hemorrhage during the introduction of the intubation tube.

New and Clark [44] expressed the opinion that radical operations, such as thyrotomy with or without cautery, should never be performed except when all other methods have failed to relieve the distressing symptoms. However, thyrotomy is rarely required for the removal of an angioma; most of these vascular growths can be treated satisfactorily under suspension laryngoscopy by destruction of the tumor by electrocoagulation. In the treatment of large simple hemangiomas and most cavernous hemangiomas, radium irradiation may be the method of choice.

Myxomatous Tumors.—Myxomatous tumors are commonly encountered in the larynx. At the clinic 58 such growths have been seen; this number does not include inflammatory or other types of tumors containing patches of myxomatous degeneration. However, it is likely that the greater number of the 58 so-called true myxomas were the result of an inflammatory process.

In 82 per cent of the cases of myxomatous tumors the patients were men; in 18 per cent they were women. This type of neoplasm occurs usually in adults. The ages of our patients varied between 25 and 66; 63 per cent of the patients were between the ages of 30 and 50.

Myxomatous tumors all have an edematous polypoid appearance (fig. 30 *A* and *B*); some are rather vascular. Clinically they cannot be distinguished from inflammatory polypoid growths (fig. 30 *C*), but this is only a matter of academic concern, since both conditions are treated in an identical manner. Usually myxomas take the form of polyps or nodules but may appear as polypoid thickenings of the vocal cords or as large pedunculated tumors. Twenty-eight of the myxomatous tumors in our series were polyps; 19 were nodules; 9 were thicken-

44. New, G. B., and Clark, C. M.: Angiomas of the Larynx: Report of Three Cases, Ann. Otol., Rhin. & Laryng. **28**:1025-1049 (Dec.) 1919.

ings, and 2 were large pedunculated masses. Although these growths may arise anywhere in the larynx, the most common site for their development is on the vocal cords. Eighty-one per cent of the 58 myxomas were attached to the true cords and usually involved their anterior two thirds; only 1 of these tumors was situated on the posterior third of a vocal cord.

Symptoms produced by these tumors depend on their size; the tumors may vary from 1 mm. to 3 cm. in diameter. Hoarseness was a symptom in all but 3 of our cases; it had been present from two

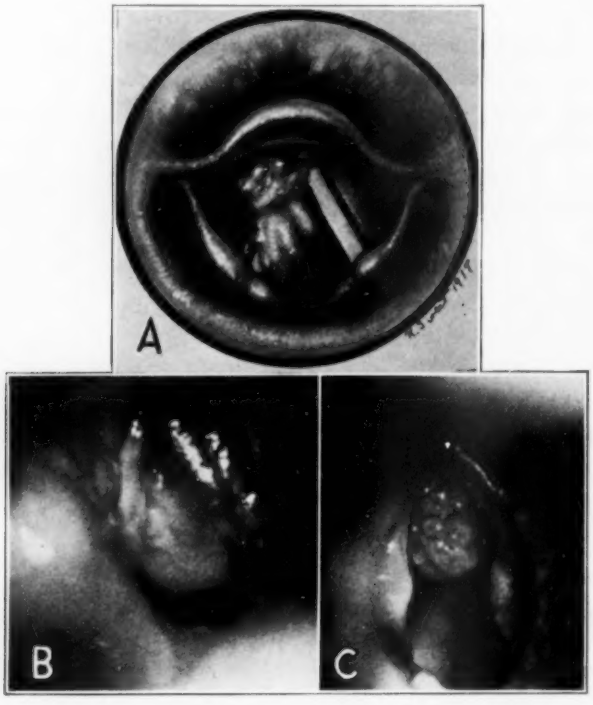

Fig. 30.—*A*, fibromyxomatous polyp. *B*, fibromyxomatous polyp (photograph). *C*, inflammatory papillary polypoid tumor (photograph).

months to twenty-two years, the average duration being two years and eight months. A slight persistent cough was a distressing symptom in 4 cases. In only 2 caes was dyspnea present; in 1 of these cases a polypoid mass completely filled the supraglottic portion of the larynx.

One patient had had her throat cut six years previously. This injury had necessitated an emergency tracheotomy, and since then she had been unable to breathe without the tracheal cannula. On laryngeal examination it was surprising to find, besides a soft cicatricial stenosis, a polypoid tumor attached to the vocal cord on the left side. This flabby

mass completely filled the larynx and extended into the esophagus. It was removed, and histologic examination disclosed that it was a mass of inflammatory fibromyxomatous polyps. Surgical correction of the stenosed larynx was to be undertaken, but the patient left the clinic and did not return.

Myxomatous tumors of the larynx are easily removed. When of small dimensions, they can be clipped off with laryngeal forceps under indirect laryngoscopic vision. However, we prefer to remove the large growths by suspension of the patient; the polypoid mass can then be clipped off and the base coagulated lightly by diathermy. As with other laryngeal neoplasms, if the myxoma is of sufficient size to cause pronounced dyspnea it is well to perform preliminary tracheotomy before treating the growth. Myxomatous tumors, when thoroughly eradicated, do not recur.

Cysts.—Cysts of the larynx are not rare, but they are not often encountered. Thirty-five laryngeal cysts have been seen at the clinic. Although such cysts have been reported as occurring at birth, the majority of them occur during adult life. Greene [45] reported 3 cases in which a cyst of the epiglottis had obstructed the glottis of a newborn infant and had resulted in the death of the infant. All of our patients with laryngeal cysts were adults; their ages ranged from 14 to 74.

As to situation of these tumors, it is generally accepted that cysts of the larynx occur most frequently on the epiglottis; this probably is due to the abundance of mucous glands in this region. However, in reviewing the cases of laryngeal lesions, we were able to find but 4 cases of cyst of the epiglottis, 2 of bilateral cysts (fig. 31 *A*). Twenty-five cysts involved the vocal cords (fig. 31 *B*); 2, the arytenoepiglottic fold; 1, the false vocal cord, and 3, the ventricle. There was 1 case of multiple cysts of the larynx (fig. 31 *C*). The anterior two thirds of the vocal cords are the common site for the development of cystomas; the posterior third was involved in only 1 of our cases. Usually embryonal cysts occur in the arytenoepiglottic fold or in the lateral wall of the larynx.

Cysts of the larynx generally offer no difficulty in diagnosis. A few possess a broad base, but the majority are pedunculated. All are soft globular compressible growths, and they are usually shiny and semitranslucent and may have blood vessels coursing over their surface. Cysts on the vocal cords have a tendency to be more translucent and grayish than those on the epiglottis, which often have a pink or reddish cast. Laryngeal cysts may be multiple, particularly when they occur about the epiglottis. As with other benign tumors, the gross appearance of some cysts is not diagnostic. In one of our cases bilateral

45. Greene, D. C., Jr.: An Unusual Cyst of the Larynx, Laryngoscope **17**:800-804 (Oct.) 1907.

small tumors were found at the junction of the anterior and the middle third of both vocal cords; these were thought to be fibrous vocal nodules but on removal proved to be mucous cysts. In another case a laryngeal cyst which was attached to the ventricle appeared to be a malignant neoplasm.

Cysts on the vocal cords usually are smaller than those elsewhere in the larynx. In our 35 cases the size of the cysts varied from 1 mm. to 4 cm. in diameter; those on the vocal cords averaged 3 to 5 mm. in diameter, while those on the epiglottis, arytenoepiglottic fold and ventricle were larger.

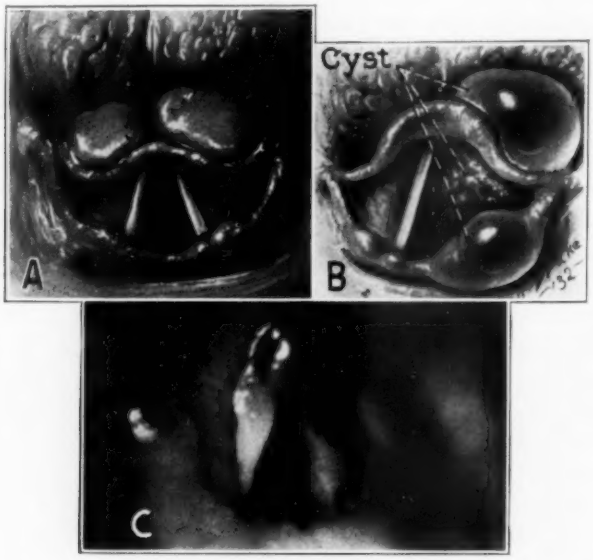

Fig. 31.—*A*, bilateral mucous cysts of the epiglottis. *B*, cyst of the left side of the larynx. *C*, hemorrhagic cyst of the anterior third of the left vocal cord (photograph).

Since cysts of the larynx usually do not reach large dimensions, dyspnea is an uncommon symptom. It was present in but 2 of our cases; in 1 of these cases the cyst was 3 cm. in diameter, and in the other case it was about 4 cm. in diameter. Hoarseness is present when the growths involve the vocal cords. Cough and pain are rare.

In 1 case a cyst of the epiglottis almost filled the hypopharynx. It was ruptured before the site of attachment could be determined. The sac, which was attached to the epiglottis, was removed.

The only cyst of particular interest in our series of cases was one of congenital origin.

Case 23.—The patient was a girl aged 14. Stridor had developed a few days after her birth. Difficult breathing and hoarseness had persisted until she was

7 years old, at which time the laryngeal cyst had been discovered and drained. For a time this had relieved her symptoms, but subsequently the dyspnea and hoarseness had returned. Since then on several occasions the cyst had been incised and drained. At the last two operations pus had been obtained by incision. Examination at the clinic revealed a hemorrhagic inflammatory mass and a draining sinus; the mass was situated in the right arytenoepiglottic fold and was about 4 cm. in diameter. Under suspension laryngoscopy the cyst was opened wide, and about 1½ ounces (44.4 cc.) of pus were evacuated. The wound healed, and the patient has had no further trouble.

In the treatment of laryngeal cysts, laryngologists have reported that puncturing of some of these growths has resulted in their disappearance. However, we recommend removal of the wall of the cyst with forceps and cauterization of the base by diathermy, visualization being obtained with the indirect laryngoscope or by suspension laryngoscopy. In some of the embryonal cysts the large size and situation may not permit proper removal under suspension laryngoscopy and thyrotomy may be necessary.

Inflammatory Tumors.—The significant position which inflammatory growths occupy in any discussion on benign tumors of the larynx is evident from the observation that 46 per cent of the benign tumors in our series of cases were inflammatory. Their importance is further magnified by the fact that clinically an inflammatory tumor may bear an exact similarity to almost any type of laryngeal neoplasm, benign or malignant, not only in gross appearance but in mode of onset and in production of symptoms.

Since inflammatory laryngeal growths undoubtedly are the result of chronic hyperemia or catarrhal inflammation of the mucous membrane or are due to traumatism in the form of laryngeal strain, misuse or overuse of the voice, it is not surprising that many patients with such growths are clergymen, singers or public speakers. Neither is it strange that men are far more frequently affected than are women, as men are more subject to laryngeal catarrh and strain than are women. It also is not odd that inflammatory tumors are rare among children, when one considers that laryngeal catarrh and traumatism probably must be present for years before an inflammatory neoplastic mass can become evident.

Three hundred and thirty-two patients with inflammatory tumors of the larynx have come to the clinic for treatment; 75 per cent of them were men. The youngest patient was 18; in fact, only 3 of the 332 patients were under 20, and 6 per cent were less than 25; 70 per cent were between 25 and 50, and 24 per cent were between 50 and 76. It may be concluded, therefore, that inflammatory tumors of the larynx occur usually in middle-aged persons. Furthermore, it would not be

amiss to state that a tumor in the larynx of an infant, of a child or of a youth is almost certain to be something other than an inflammatory growth.

Symptoms produced by inflammatory tumors of the larynx are those of benign laryngeal tumors in general. Impairment of the quality of the voice or actual hoarseness is common to all inflammatory growths. Dyspnea occurs occasionally; pain and cough occur but rarely.

After an analysis of our 332 cases, we have classified inflammatory laryngeal growths into the following six groups in accordance with their clinical appearance: (1) fibrous nodules; (2) polypoid tumors; (3) pedunculated nonpolypoid tumors; (4) papillary tumors; (5) inflammatory thickenings, and (6) contact ulcer granulomas.

Fibrous Nodules: The term "fibrous nodules" refers to the clinical aspect of these tumors rather than to their histologic appearance. Histologically, these tumors may be diagnosed as simple inflammatory tissue, hemorrhagic inflammatory tissue, vascular inflammatory tissue (pseudoangiomas) or fibrous inflammatory tissue (subepithelial fibrosis and inflammation).

Fibrous nodules occur on the vocal cords, appear firm and fibrous rather than polypoid and are usually covered with normal mucous membrane. They vary from 0.5 to 8 mm. in diameter and average about 3 mm. The middle third of the vocal cord is involved most frequently; the junction of the anterior and the middle third is involved next most commonly, and the anterior third of the vocal cord is involved least often. The posterior third is but rarely the site of a nodule; only 2 of 96 fibrous nodules seen at the clinic occurred on the posterior third of a vocal cord. Usually the lesion is single, but the involvement may be bilateral, as it was in 15 of our 96 cases. Fibrous nodules have a tendency to be bilateral among singers and public speakers who have misused their vocal organs. In such instances the nodules represent inflammatory changes in the vocal cords owing to singing or speaking out of one's normal vocal register or attempting to produce a volume of tone beyond one's normal capacity.

When they are small fibrous nodules may not cause any apparent symptoms. Twelve of our patients were entirely unaware of the presence of such a growth or growths and complained of no symptoms whatsoever. However, nodules on the vocal cords usually cause much hoarseness.

For treatment of fibrous nodules on the vocal cords we recommend their removal in most cases. Merely clipping off the lesions superficially under suspension laryngoscopy or with indirect laryngoscopic vision will result in cure. However, in cases of singers and public speakers who have misused their voices, it is important to remember that the nodules on the vocal cords are not the direct cause of the

hoarseness or inability to speak or sing clearly but rather are the result of a misused and injured vocal mechanism. In short, the nodules represent just one development in a series of changes that occur in a vocal cord which has been misused. Removal of the nodules may overcome some of the hoarseness, but it will not improve the quality of the singing or speaking voice. Particularly if the patient is a professional singer, the nodules should be left entirely alone, lest the patient later decide that the surgical removal of the nodules was the cause of injury to his or her voice. Rather than operation in such cases, a conservative form of treatment should be adopted; this should consist of vocal rest and measures directed toward the correction of the patient's method of singing.

Polypoid Tumors: The most common of all inflammatory growths in the larynx are polypoid tumors. At the clinic 168 such tumors have been observed; 101, or 60 per cent, of these tumors were nodular; 52, or 31 per cent, were polypoid, and 15, or 9 per cent, were large and pedunculated. A special consideration of these tumors is unnecessary, since they are identical with myxomatous laryngeal neoplasms, which have been thoroughly described. Myxomatous and polypoid inflammatory tumors are alike in gross appearance, in mode and time of onset and in production and duration of symptoms. Both types of lesion are treated in a similar manner. In fact, the two conditions are indistinguishable except on microscopic examination. However, in our series of cases, inflammatory polypoid tumors occurred three times more frequently than did myxomatous growths.

Pedunculated Nonpolypoid Tumors: Pedunculated nonpolypoid inflammatory tumors are firm growths attached to the larynx by a narrow pedicle. They do not undergo ulceration. Except for the pedicle, they are similar to fibrous nodules. We have seen 11 patients, all men, with such pedunculated tumors. The growth was small, except in 1 case, in which it was about 1 cm. in diameter and was attached to the anterior commissure. During inspiratory and expiratory efforts this growth flopped up and down between the vocal cords and caused a moderate amount of dyspnea. In another case the growth was attached in the interarytenoid region, an unusual situation for the development of a benign tumor. Methods used in the treatment of these pedunculated inflammatory tumors were similar to those employed in the removal of fibrous nodules.

Papillary Tumors: Papillary inflammatory tumors include a group of inflammatory growths which may appear as single papillary lesions, as nodules with a papillary surface or as warty thickenings. Seventeen cases of papillary tumors were observed in our series of cases of benign laryngeal tumors. When they occur as single papillary lesions they cannot be distinguished from ordinary single papillomas. Similarly,

papillary inflammatory nodules appear to be clusters of papillomas. In 15 of our 17 cases the lesions were diagnosed clinically as papillomas.

In 1 case examination revealed multiple warty papillary lesions involving the entire right false cord, the interarytenoid region and the posterior aspect of the epiglottis. Biopsy disclosed that the lesions consisted of simple inflammatory tissue undergoing hyaline degeneration. Under suspension laryngoscopy the growths were destroyed by electrocoagulation.

The treatment of papillary inflammatory tumors is the same as that used for the foregoing types of inflammatory tumors. When papillary tumors are extensive they can be easily destroyed by diathermy.

Inflammatory Thickenings: An inflammatory process may produce a tumor-like thickening of a part of the larynx rather than a nodular, polypoid, pedunculated or papillary type of tumor that projects into the laryngeal lumen. We refer to this tumor-like thickening as an "inflammatory thickening." Twenty-one cases of inflammatory thickening of the larynx have been observed at the clinic. The patients were between 20 and 65 years of age; 11 were men, and 10 were women.

These tumors usually involve the true or false vocal cords or the lateral walls of the subglottic region. Occasionally only a small portion of a true or false vocal cord is affected, but the entire cord often is involved. Sometimes the condition is bilateral. When of small dimensions the lesions produce few symptoms other than hoarseness, but when large they may cause sufficient obstruction in the larynx to induce severe dyspnea. If such a tumor involves a vocal cord extensively, the cord may become fixed as a result of inflammatory induration. A fixed cord, together with, perhaps, some inflammatory ulceration, is strongly suggestive of a malignant condition. Without fixation of a cord, if the thickening is associated with other inflammatory changes in the larynx, there is usually little difficulty in determining the inflammatory character of the tumor.

Many of these lesions are associated with a subacute inflammatory process, and they will often disappear spontaneously. However, they are frequently indolent, requiring weeks or even months to undergo resolution. Thickenings of a chronic inflammatory nature usually do not disappear and require removal.

Before treatment of an inflammatory thickening of the larynx is undertaken biopsy should be done to determine beyond a doubt that the condition is inflammatory. If it is due to a chronic inflammatory process, the lesion may be destroyed by diathermy under suspension laryngoscopy. However, for the subacute forms of inflammatory thickening a nonsurgical conservative form of treatment should be instituted. Rest of the voice should be prescribed, and inhalations of steam

and oil sprays used. When the growth is of sufficient size to produce marked respiratory obstruction, tracheotomy should be performed to relieve the dyspnea.

Since these inflammatory thickenings clinically are often mistaken for malignant or other neoplasms, they are of sufficient importance to warrant a brief review of 10 of our cases, which are interesting from a diagnostic point of view.

CASE 24.—A man aged 37 had a thickening of the anterior third of the right false cord. This condition was thought to be malignant, but biopsy proved that the lesion was inflammatory. It was destroyed by electrocoagulation.

CASE 25.—A man of 50 had an extensive thickening of the entire right vocal cord, which was fixed. Although repeated biopsy failed to disclose evidence of malignancy, the condition was thought to be malignant, and thyrotomy was performed. However, the tumor was found to be inflammatory throughout.

CASE 26.—A woman of 50 had bilateral inflammatory thickening of both vocal cords. This caused much dyspnea, and tracheotomy was necessary. Biopsy disclosed that the lesion was inflammatory, and no further treatment was undertaken. The condition required about a year to disappear, and there was considerable scarring in the larynx.

CASE 27.—A woman of 49 had bilateral subglottic masses. They caused much obstruction to the trachea, and tracheotomy was necessary. The tumors were thought to be inflammatory, and this opinion was confirmed by microscopic examination of a piece of the growth. No other treatment was given. The patient left the clinic and did not return.

CASE 28.—A man of 57 had smooth nodular thickening of both vocal cords. A diagnosis of amyloidosis was made, but biopsy proved that the lesions were entirely inflammatory. The patient did not stay at the clinic for treatment.

CASE 29.—A man of 48 had a thickening of the posterior half of the right vocal cord. The lesion was thought to be malignant, but biopsy disclosed that it was inflammatory. He was given conservative treatment, and the lesion healed in seven months.

CASE 30.—A man of 65 had a thickening of the anterior third of the right vocal cord and some limitation of movement of the cord. It was thought that the lesion possibly might be malignant, but biopsy proved it to be inflammatory. The lesion was destroyed by diathermy under indirect laryngoscopic vision.

CASE 31.—A woman of 45 had a granulomatous-appearing thickening of the anterior half of the left vocal cord and of the anterior commissure. The clinical diagnosis was tuberculosis or an indeterminate granuloma, but the thickening was seen on histologic examination to consist of simple inflammatory tissue. The entire lesion was destroyed by electrocoagulation.

CASE 32.—The patient was a woman of 34. Examination disclosed bilateral uniform swellings on the lateral walls of the larynx in the subglottic region. A clinical diagnosis of laryngeal amyloidosis was made, but microscopic examination disclosed that the tumors were composed of simple inflammatory tissue. Tracheotomy was performed to relieve the dyspnea. This was followed by light irradiation of the tumors, which disappeared in six months.

CASE 33.—The patient was a woman of 20. Examination revealed a solid-appearing rounded tumor attached to the entire right vocal cord. Biopsy dis-

closed that the tumor was composed of inflammatory tissue. The patient did not stay at the clinic for treatment.

Contact Ulcer Granulomas: We have observed 19 cases in which a laryngeal granuloma was associated with a contact ulcer. In 1 case bilateral granulomas were present. The term "contact ulcer" was introduced by Chevalier Jackson to designate the inflammatory ulceration which occasionally occurs over one or both vocal processes in the larynx. It is thought that these lesions are due to chronic laryngitis associated with vocal abuse.

The ulcer itself is superficial and frequently causes the development of a granulomatous mass, which, as we have found, may vary from 2 or 3 mm. to 1.5 cm. in diameter. Often this condition causes pain or laryngeal discomfort. Four of our patients complained of pain. Hoarseness is frequently present, owing to the associated laryngitis or to mechanical interference by the granuloma with the movements of the vocal cords. In 1 case dyspnea was caused by a granuloma which partially obstructed the glottis.

Contact ulcer granulomas are not difficult to diagnose, although they occasionally are confused with tuberculosis or epithelioma.

Two important measures are necessary in the eradication of this type of granuloma: (1) removal of the growth and the associated ulceration and (2) enforcement of complete vocal rest until the wound over the vocal process has healed entirely. Frequently without vocal rest the granuloma will recur, and it sometimes will return in spite of persistent silence. In treating our patients, after removal of the growth we insist on absolute vocal rest until the affected region has healed, and we inform the patient at the start that the granuloma may recur in spite of the treatment.

Xanthomas.—Few cases of xanthoma of the larynx have been reported. Undoubtedly many patients with dermal xanthomas also have similar laryngeal lesions, which escape notice. In consequence, the number of reported cases of xanthoma of the larynx is not a fair estimate of the incidence of the disease.

Only 4 cases of laryngeal xanthoma have been observed at the clinic. A detailed review of the first 2 of these was published by Finney, Montgomery and New [46] in 1932, and the other 2 were reported by New [47] in 1925.

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46. Finney, W. P.; Montgomery, H., and New, G. B.: Xanthoma Multiplex: Two Cases Involving the Larynx and Trachea, and Associated with Diabetes Insipidus, J. A. M. A. **99**:1071-1074 (Sept. 24) 1932.

47. New, G. B.: Xanthoma of the Pharynx and Larynx, Arch. Otolaryng. **22**:449-453 (Oct.) 1935.

Xanthomatous lesions in the larynx as well as in the mouth and pharynx appear as nodules or plaques of a yellowish color (fig. 32). The nodules have a smooth and sometimes a nodular surface. At times they grow sufficiently large to obstruct, at least partially, the laryngeal lumen. As previously stated in the consideration of the pathologic picture, these tumors go through an inflammatory stage, an intermediate tumor stage and finally a stage of fibrosis. In the regressive phase enough scarring may develop to cause serious interference with the vocal mechanism or to produce stricture or contracture of the larynx. Xanthomatous infiltrations of the upper part of the respiratory tract are practically always associated with similar lesions in the skin.

CASE 34.—A man aged 27 had many xanthomatous infiltrations in the skin and scattered elevated lesions on the palate, tonsillar pillars and posterior pharyngeal wall, which extended down over the epiglottis. There was marked scarring

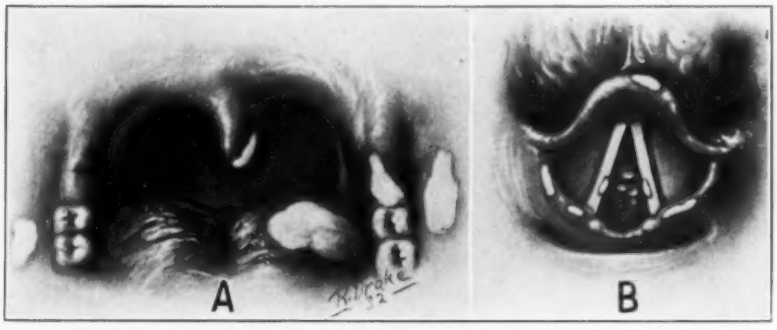

Fig. 32.—*A*, xanthomatous lesions of the mouth and pharynx. *B*, xanthomatous lesions of the larynx.

of the pillars and of the soft palate, which resulted in a nasopharyngeal stricture. The epiglottis was scarred, contracted and drawn downward; therefore, a view of the larynx was impossible even when an attempt was made to retract the epiglottis. Owing to this scarring and contracture of the vestibule of the larynx, tracheotomy was necessary to relieve the associated dyspnea.

CASE 35.—The patient was a man aged 42. Besides large confluent bronze-colored nodules in the skin, he had multiple small lesions in the mouth, larynx and trachea. In the larynx there were 8 xanthomatous infiltrations, which involved the epiglottis, the arytenoepiglottic folds and the vocal cords.

CASE 36.—The patient was a woman of 48. Except for a small nodule on the back of the right shoulder and one in the left midcervical region, there was no involvement of the skin. Examination of the pharynx revealed extensive yellowish deposits on the anterior pillars of the tonsils, on the palate, in the hypopharynx and in the vallecula epiglottica. There was one small nodule in the subglottic region, below the anterior commissure. Both of the arytenoid cartilages appeared to be scarred and to be pulled forward.

CASE 37.—A woman aged 46 came to the clinic because of hoarseness, which had been present for eight months. She had no dermal lesions, and there were no

xanthomas in the mouth or pharynx. Examination of the larynx revealed a rounded slightly nodular yellowish nonulcerated tumor, which involved the posterior three fourths of the true and false vocal cords. There was slight limitation of movement of the left cord. A clinical diagnosis of xanthoma was confirmed by histologic examination.

In most instances treatment of xanthomas in the larynx or elsewhere about the body is unsatisfactory. The nodules or plaques may be excised or destroyed by diathermy, but new growths are likely to develop. In a few cases the lesions have been known to disappear spontaneously. In cases 34 and 35 treatment consisted of roentgen therapy and a diet low in calories and fat. However, such treatment had no effect on the lesions. In case 36 no treatment for the xanthomas was instituted, because the woman had pernicious anemia, the treatment of which was considered of more importance than therapy directed to curing the xanthomas. The tumor in case 37 was removed by thyrotomy, with an excellent curative and functional result.

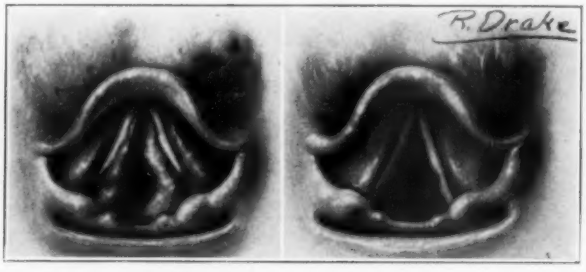

Fig. 33.—Larynx before and after the removal of bilateral amyloid tumors.

When marked scarring in the larynx is present, tracheotomy may be necessary to relieve the dyspnea.

Amyloid Tumors.—Amyloid tumors of the larynx are uncommon; only 18 cases have been observed at the clinic. These tumors may appear as a diffuse subepithelial infiltration, as a localized tumor or as a degenerative process in a preexisting tumor.

In our series the youngest patient was 30, and the oldest was 68. Thirteen of our patients were between 38 and 55. Ten of the 18 patients were men, and 8 were women.

Most of our patients had been aware of the presence of a laryngeal growth for several months before they came to the clinic. Although 1 patient had noticed hoarseness for only two days and another had had persistent hoarseness for ten years, the duration of symptoms averaged about two years and ten months. The symptoms vary with the size and situation of the amyloid deposit. All of our patients complained of hoarseness, even though the growth did not involve the vocal cords in some instances. In 6 cases the growth was large enough to cause dyspnea. Cough and pain were rare.

On laryngeal examination amyloid tumors are found to be either nodular growths, sometimes pedunculated, or infiltrative swellings or masses (figs. 33 and 34). The nodular amyloid tumors in our series of cases were all attached to the true vocal cords in their anterior two thirds. In 1 case bilateral amyloid tumors which looked much like the nodules which involve the vocal cords were present. Usually the amyloid nodules have a smooth surface and possess a reddish color. Occasionally they are polypoid. They vary from 1 mm. to 1 cm. in diameter. The subepithelial amyloid infiltrations appear as smooth or irregular thickenings. Most frequently these thickenings involve the true or false vocal cords or the lateral walls of the subglottic region. In many cases subepithelial amyloid infiltrations consist of bilateral tumors in the larynx. Occasionally the infiltrative process involves

Fig. 34.—Bilateral amyloid tumors of the larynx.

the entire circumference of the larynx or trachea, as occurred in 1 of our cases. In such a case dyspnea may become a grave symptom.

The nodular or pedunculated amyloid tumors are easily removed by clipping off or by electrocoagulation under suspension laryngoscopy. However, in the large infiltrative types, thyrotomy is necessary to gain adequate exposure for removal. When the amyloidosis involves the circumference of the larynx, its removal by thyrotomy will result in much postoperative scarring, which may necessitate the permanent use of a tracheotomy tube. Such a procedure may be advisable, in spite of the subsequent scarring, to prevent the amyloid infiltrations from extending down the trachea.

Epithelial Hyperplasia and Leukoplakia.—Twenty-three cases of epithelial hyperplasia and 10 cases of leukoplakia have been encountered at the clinic. The latter group does not include the many cases in which leukoplakia was associated with epithelioma. Both of these conditions are of great importance because of their potential malignant

qualities. To emphasize this fact, we wish to call attention to 3 cases of leukoplakia. Clinically, the lesions in these cases appeared to be perfectly benign, but microscopic examination disclosed squamous cell epithelioma, grade 2.

In cases of epithelial hyperplasia and leukoplakia the ages of the patients varied from 27 to 74. With but 2 exceptions, hoarseness occurred in all cases. Cough and expectoration were frequent symp-

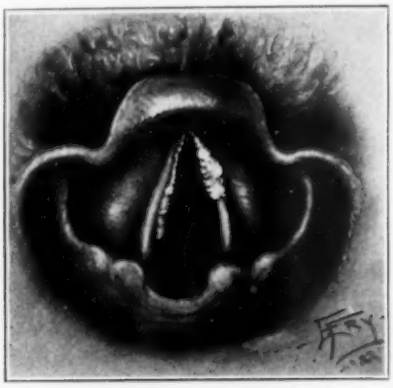

Fig. 35.—Leukoplakia involving both vocal cords.

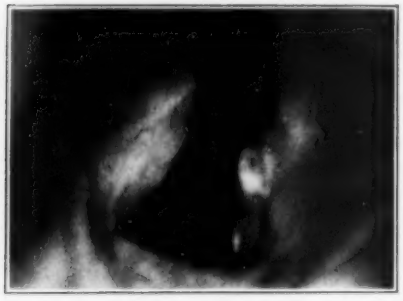

Fig. 36.—Epithelial hyperplasia over a contact ulcer at the base of the right vocal cord (photograph).

toms. Since these lesions rarely become large enough to obstruct the larynx, dyspnea is an exceptional symptom.

Leukoplakia is rather constant in its appearance. In all our cases the lesions were confined to the true vocal cords and appeared as whitish patches in the mucous membrane. Those which were very thick had a roughened or papillary aspect (fig. 35).

Epithelial hyperplasia (fig. 36) is extremely variable in its appearance. Often the condition appears similar to leukoplakia, but in a few of our cases the lesions were polypoid. Epithelial hyperplasia may

acquire a papillomatous appearance or may form nodular growths. Clinically, in some of our cases the lesions were thought to be fibrous nodules or papillomas. Usually, however, epithelial hyperplasia occurs as a flat thickened whitish patch which resembles leukoplakia. Many of these lesions can be definitely diagnosed only by microscopic examination. Although they may be situated anywhere in the larynx, the majority of such growths occur on the vocal cords. This was true in all our cases except 1 in which the growth involved the right arytenoid cartilage and 1 in which the lesion was situated on the epiglottis. Epithelial hyperplasia is often a secondary finding in the mucous membrane, covering other types of benign tumors.

Of the utmost importance in the treatment of these two conditions is a thorough microscopic examination of the lesions. Some of the small epithelial hyperplastic growths may be clipped off and the bases electrocoagulated. However, in the majority of cases of epithelial hyper-

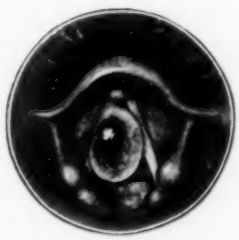

Fig. 37.—Prolapse of the left laryngeal ventricle.

plasia, and particularly in cases of leukoplakia, we prefer to suspend the patient and remove the lesion for histologic examination. The base of the lesion then is destroyed by diathermy. If a small low grade epithelioma is found in situ and if the involved region can be clearly visualized, we feel that it is justifiable to destroy the entire growth by electrocoagulation under suspension laryngoscopy rather than resort to thyrotomy. However, in all cases in which a malignant lesion is found, it should be removed by thyrotomy. Even in cases of thickened leukoplakia, if adequate exposure cannot be obtained under suspension laryngoscopy, thyrotomy may be advisable.

Prolapse of the Laryngeal Ventricle.—The 6 patients with prolapse of the ventricle whom we have seen were between 27 and 71 years of age. Two of the cases were reported by New [48] in 1915.

All the patients had had hoarseness for three months to three years. Constant cough and expectoration were distressing symptoms in 2 cases.

48. New, G. B.: Prolapse of the Ventricle of the Larynx, Laryngoscope **25**: 145-147 (March) 1915.

None of the patients were aware of any shortness of breath, although it is conceivable that the prolapsed tissue could be of sufficient size to cause dyspnea.

These growths are usually found in the anterior third of the larynx, although the entire length of the ventricle may be involved. The prolapse may be bilateral, as it was in 1 of our cases. In 3 cases the prolapse involved the right ventricle, and in 2 cases it involved the left ventricle. Clinically, the prolapsed tissue appears as a smooth grayish pink round tumor, emerging from the ventricle between the true and the false vocal cords (fig. 37). It can be tucked back into the ventricle with a probe. In 1 of our cases after the tissue had been replaced the voice became clear, but the prolapse recurred when the patient coughed.

After one has observed a case of prolapse of the ventricle, there is little possibility of confusing the condition with true benign tumors of the larynx.

Attempts to contract the prolapsed tissue by the application of caustic solutions to produce inflammation and subsequent scarring usually result in failure. We recommend complete elimination of the prolapse with biting forceps, a snare or a scalpel, or by diathermy, visualization being obtained with the indirect laryngoscope or by suspension laryngoscopy.

NASAL TEETH

REPORT OF A CASE

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PHILADELPHIA

This paper consists of (1) a brief discussion of teeth found in the nose, (2) the report of a case and (3) a concise bibliography. A discussion and a bibliography are included because of the lack of either in the literature available in the English language.

The German poet Goethe,[1] in 1797, was the first to report a case of a tooth in the nose. Gassner [2] in 1918 found reports of 70 cases in the literature, and Kramer [3] in 1932 found 100. At least 4 other cases have been reported since, and there are many which have not been reported.[4]

Most nasal teeth are maxillary first incisors; some are canines, and a few are premolars and molars. Deciduous teeth are found, but not often. It is unusual to find more than one tooth in the nose. Probably a little more than one half of the teeth so found are supernumerary.

The cause is obscure.[5] Hereditary influences probably play a part, though not an important one. Several cases of anomalies of the teeth occurring in successive generations have been reported, but none in which there were nasal teeth. Interruption of the development of the embryo at certain critical stages, according to Crosby,[6] tends to produce accessory budding, and hence supernumerary teeth. Stafne [7] and others

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Read at a meeting of the Section on Otolaryngology of the College of Physicians of Philadelphia, Oct. 20, 1937.

1. Heinemann, K.: Goethe's Werke, Leipzig, Bibliographische Institut, 1900, vol. 17, p. 151.

2. Gassner, O.: Ueber das Vorkommen von Zähnen in der Nasen- und Kieferhöhle, Zahnärztl. Rundschau **27**:89 (March) 1918.

3. Kramer, A.: Zur Frage der retinierten und ektopischen Zähne, Inaug. Dissert., University of Marburg, 1932.

4. Ivy, R. H.; Ennis, L. M., and others: Personal communications to the author.

5. Heyng, F.: Ein Beitrag zu den sogenannten Nasenzähnen, Inaug. Dissert., Rheinischen Friedrich-Wilhelms-Universität, Bonn, October 1932. Wegner, H.: Nasenzähnen und Pseudo-Nasenzähnen, Inaug. Dissert., Ernst Moritz, Arndt-Universität, Greifswald, 1935.

6. Crosby, A. W.: To What Definite Causes May Malocclusion and Its Attending Oral and Facial Abnormalities Be Attributed? Internat. J. Orthodontia **18**:1259 (Dec.) 1932.

7. Stafne, E. C.: Supernumerary Teeth, Dental Cosmos **74**:653, 1932.

discussed the origin of supernumerary teeth; whatever their origin, their presence is an important factor in the production of nasal teeth. The incidence of nasal teeth in persons having supernumerary teeth as compared with the incidence in those who have none is at least 100: 1.

Whether or not the dental germ is supernumerary, its inversion or displacement upward will tend to cause its eruption into the nose. It is thought that the union of the two sides of the maxilla may cut off and displace upward the odontogenic epithelium. Congenital, and sometimes tertiary, syphilis, tuberculosis and rickets have been mentioned as being causative. Downs [8] stated that endocrine imbalance plays an important part. He cited cases to show that dental anomalies occur more frequently in persons with such an imbalance than in normal persons. Harelip and cleft palate were present in several cases when nasal teeth were found.

Obstruction to eruption downward may be the cause. The jaw may be too narrow; there may be a persistent deciduous tooth, or an exceptionally hard bone below may prevent the tooth from erupting normally. A scar from a previous extraction may act similarly. In several cases a previous trauma to the region has been considered causative.

Sometimes the root of an unusually long tooth projects into the nose. Rarely, a tooth that has previously erupted into the maxillary antrum erodes its way through the lateral nasal wall,[9] or a cyst containing a tooth ruptures into the nose.[10]

The symptoms most commonly encountered in cases of nasal teeth are the following: (1) unilateral obstruction to breathing, (2) chronic nasal discharge, often foul smelling, (3) crusting and (4) localized ulceration. Many others have been reported. Recurrent epistaxis occurred in 1 case [11] and conjunctivitis in another [12] and a fistula into the mouth was present in several. Headache and pain in the nose and in the face, sometimes simulating tic douloureux, are not infrequently encountered. Ennis [4] told me of 2 cases in which such symptoms were present and the condition remained undiagnosed for a considerable time.

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8. Downs, W. G., Jr.: Studies in Causes of Dental Anomalies, Genetics **12**:570 (Nov.) 1927. (Reprint, 570, Nov. 12, 1927.)

9. Marshall, J. S.: A Superior Right Wisdom Tooth Discharged from the Nasal Passages, with Remarks, Arch. Dent. **3**:279, 1886; J. A. M. A. **7**:516 (Nov. 6) 1886. Abercrombie, P. H.: Eruption of Canine Tooth into the Nasal Fossa Attended by Rhinitis Caseosa, J. Laryng. & Otol. **40**:586 (Sept.) 1925.

10. Wilms: Zahn im Nasenseptum, Deutsche Monatschr. f. Zahnh. **25**:725, 1907.

11. Carco, P.: Su un raro caso di dente soprannumerario sporgente in una fossa nasale, Arch. ital. di otol. **43**:548 (Sept.) 1932.

12. Bisi, H., and Mainini, C.: Diente heterotópico de la fosa nasal, Rev. Asoc. méd. argent. **46**:1480 (Nov.) 1932.

Fig. 1.—Roentgenogram showing a misplaced canine tooth in the floor of the nose of a man aged 20 (case of Dr. Robert Ivy). No symptoms were present.

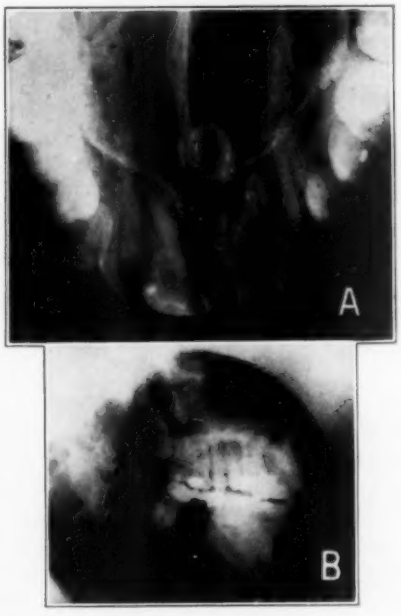

Fig. 2.—*A*, occlusal view showing a tooth apparently in the nasal floor (case of Dr. Leroy Ennis). The patient was a man aged 28. He had suffered from neuralgic pains in the face for several years. *B*, lateral view of the nasal tooth shown in *A*, indicating that the tooth was well above the nasal floor. It was embedded in the septum.

Unusual symptoms, most of which can be explained only by reflex irritation, are asthmatic attacks, nervousness, laryngospasm, otalgia, epileptiform seizures and hysteria. Occasionally there are no symptoms at all, several patients having gone to the physician only because the tooth protruded from the nostril. In the case here reported, I do not think that the symptoms were due to the presence of the nasal tooth.

The diagnosis is usually not difficult, but may be missed most easily when the tooth is almost completely embedded in the nasal mucosa and is on the nasal floor near the vestibule, where it may be overlooked in the routine examination. This has occurred a number of times. Once located, the tooth will be found to be hard, usually smooth and slightly movable when tested with a probe. This differentiates it from exostoses and odontomas, which are not movable, and from foreign bodies and bony sequestrums, which are usually readily movable. In addition, bony sequestrums are most often rough and are apt to be multiple. Cysts are usually soft. Absence of a tooth from its proper place is suggestive. Roentgen examination will make the diagnosis certain. Both a lateral and an occlusal (bird's eye) view should be taken if there is any doubt, for a tooth in this region is at times difficult to visualize and to localize. In 1 case an unnecessary operation was performed on the palate before the exact location of the tooth was determined (fig. 2). If the tooth is a retained one it will be of normal form, and if it is supernumerary it possibly will be peg shaped.

The treatment is always surgical removal. It may be necessary to make an incision in the nasal mucosa. The tooth is then removed with the forceps or the snare. In 1 case, that reported by Bisi and Mainini,[12] an operation similar to a modified Denker procedure was done. In every case I could find, the removal was followed by complete cessation of all symptoms, and in none were there any ill results.

REPORT OF CASE

W. G., a white man aged 28, single, came to the nose and throat dispensary of the Germantown Hospital. His chief complaint was frequent colds. Examination of the nose revealed a septum that was markedly deviated to the left. He had never been able to breathe well through that side of the nose. There was no history of nasal injury. The past history and physical examination showed nothing of importance. A submucous resection was performed. During the operation, after most of the deformity had been corrected, a small white object was noticed protruding slightly from the floor of the left nostril, ½ inch (1.2 cm.) posterior to the vestibule. It was hard and was slightly movable when tested with a probe. The object was easily removed with a snare, and proved to be a peg-shaped tooth. It measured ¾ by ¼ inch (1.8 by 0.6 cm.). It tapered somewhat at either end. One third had been buried in the nasal mucosa. The crown, which was exposed, pointed dorsally and a little superiorly. There was no evidence of inflammation or

ulceration about it, nor was there any visible defect in the hard palate. All the natural teeth were in place and were apparently normal. A roentgenogram showed no deformities or other supernumerary teeth. The Wassermann reaction was negative. There was no history of venereal disease, tuberculosis or rickets, and the family history was irrelevant. Dr. L. G. Grace, of the Germantown Hospital, declared that the specimen was a peg-shaped supernumerary tooth, and Dr. H. R. Churchill, of the Evans Institute, added that it did not fall under any classification of normal human teeth and should be regarded as a supernumerary specimen. There were no postoperative complications.

Dr. Ivy and Dr. Leroy Ennis furnished the roentgenograms.

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MALIGNANT DISEASE OF THE EAR

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In the series of 8 cases to be reported there were 7 of carcinoma and 1 of myxosarcoma. The 7 patients with carcinoma were all elderly; the patient with myxosarcoma was 4 years old. One patient had a carcinoma of the nose as well as of the ear. In all cases the disease was in a late stage, and either surgical treatment or irradiation or both had been given in some other hospital before the patient was admitted to the Colorado General Hospital.

GENERAL DATA

Age.—The ages ranged from 4 years to 83. (The ages were 4, 63, 66, 66, 67, 79, 80 and 83.) All the patients with carcinoma were past 60. The 4 year old child had myxosarcoma. The average age for all 8 was 63½. The average age for the 7 with carcinoma was 72.

In Robinson's [1] series of 24 cases the youngest patient was 28 and the oldest 93. The average age was 66.

In Broders' [2] series of 63 cases the average age was 61¼.

Sex.—There were 7 males and 1 female. Malignant disease of the head and neck is much more frequent in the male according to the reports of nearly all large hospitals in this country and abroad.

In Robinson's [1] series of 24 cases there were 19 males and 5 females.

In Broders' [2] series of 63 cases 76.19 per cent of the patients were males and 23.80 per cent were females.

Occupation.—Two patients were farmers; 2 were retired; 1 was a commission man; 1 was a miner; the woman did housework, and the child was only 4 years old. The former occupation of 1 of the retired men was not given. The other had worked on a farm, at plastering and at stone masonry.

From the Department of Otolaryngology, University of Colorado School of Medicine and Hospitals.

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 16, 1938.

1. Robinson, G. A.: Malignant Tumors of the Ear, Laryngoscope **41**:467-473, 1931.

2. Broders, A. C.: Epithelioma of the Ear: A Study of Sixty-Three Cases, Tr. Am. Otol. Soc. **15**:402-406, 1921.

In Broders'[2] series of 63 cases 64.44 per cent of the male patients were farmers.

Biopsy.—A biopsy was performed for 6 of the 8 patients. The first 2 did not have a biopsy because they were not in the hospital long and the malignant disease was so far advanced that a biopsy was not advised.

Wassermann Test.—The Wassermann test was made for 4 of the 8 patients. The other 4 probably remained in the hospital too short a time for the laboratory staff to make the test. The 4 tests made were negative. The history and physical examination of the 4 patients not tested indicated that their tests, too, would very likely have been negative.

Irradiation.—In all but 1 case (case 2) irradiation was given in another hospital before admission, in the Colorado General Hospital or after discharge. The patient in case 2 was sent to the hospital for irradiation, but he died of pneumonia the fifth day after admission, at the age of 83. He had pneumonia and was irrational the day he was admitted. Surgical intervention supplemented by irradiation, in the opinion of most of the best otolaryngologists, gives the best result.

SURVEY OF THE LITERATURE

Histologic Differentiation.—In the present series, of the cases of proved carcinoma the disease was of the squamous cell type in all except 1, in which it was of the basal cell type.

Lukens,[3] Schall,[4] Furstenberg,[5] Fraser[6] and Thorell[7] have all reported adenocarcinoma of the external ear. Lukens[3] stated the belief that adenocarcinoma is more common than the rare reports in the literature indicate and that if biopsy were done of all tumors of the ear more would be known about its frequency. Adenocarcinoma has been the least frequently reported of all the malignant tumors of the ear.

Lukens'[3] treatment, termed "radio-ionic medication of uranium," is unusual, especially for a patient who refused surgical intervention.

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3. Lukens, R. M.: Adenocarcinoma of the External Auditory Canal, Ann. Otol., Rhin. & Laryng. **45**:567-573, 1936.

4. Schall, L. A.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **22**:548-553 (Nov.) 1935.

5. Furstenberg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **33**:677-689, 1924.

6. Fraser, J. S.: Malignant Disease of the External Acoustic Meatus and Middle Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **23**:71-77, 1930.

7. Thorell, I.: Treatment of Malignant Tumors of the Middle Ear at Radiumhemmet, Stockholm, Acta radiol. **16**:242-253, 1935; abstracted, Bothman, L.; Brown, E. V. L., and Shambaugh, H. E.: Year Book of Eye, Ear, Nose and Throat, Chicago, Year Book Publishers, Inc., 1935, p. 358.

Denison [8] has reported prickle cell epithelioma of the ear with involvement of the facial nerve in the tympanum. He classified carcinoma into the following groups:

1. *Squamous or Flat Cell Cancers:* These arise in the skin, esophagus, etc., or wherever there is stratified epithelium, and are known as epitheliomata.

2. *Basal Cell Cancers:* These arise chiefly in the skin, but analogous tumors are found elsewhere. They are relatively non-malignant and are the basis of the so-called rodent ulcers.

3. *Cylindrical Cell Cancers:* These are analogous to the polyps or polypoid adenomata of mucosae which have glands lined with columnar epithelium. They retain the tendency to form gland-like structures, lined with cylindrical epithelium and are hence called adenocarcinomata.

4. *Cancers Derived from Acinous Glands.*

He said: "Prickle cell epitheliomata belong to the squamous cell type."

Malignant tumor of the ear is rare, according to most authors. Fraser,[6] at the Royal Infirmary at Edinburgh, Scotland, found only 13 cases among 6,600 cases of external disease of the ear. Robinson [1] found only 1 case of malignant disease among 4,000. Broders [2] reported 33 per cent were of the basal cell type, 61 per cent of the squamous cell type and 5 per cent melanotic, in a series of 63 cases of malignant tumor. Eczema, trauma, frostbite and keratosis favor the development of malignant disease. Robinson [1] listed eczema, sebaceous cyst, papilloma, senile keratosis, trauma, frostbite, chronic mastoiditis with polyps in the external canal and metastasis from other organs as causes.

Broders [2] has reported 63 cases of epithelioma of the ear. He found three types of tumor as follows: (1) basal cell epithelioma, or rodent ulcer; (2) squamous cell epithelioma; (3) melanoepithelioma. He said:

> While all of these epitheliomas are cancers there is a great difference in the effect they produce on the patient. It is well known that basal-cell epithelioma is the least malignant and melano-epithelioma the most malignant of the three; the squamous-cell epithelioma is between the two.

Of Broders' [2] patients "nineteen and four hundredths per cent had a family history of malignancy." A "history of a previous wart, mole, scab, keratosis, ulcer, eczema, pimple, or lump, at the site of the lesion" was elicited from 63.49 per cent. A history of injury or infection was obtained from 19.04 per cent.

Guttman [9] has reported a case of carcinoma of the ear in which the patient, a man of 60, had had acute suppurative otitis media five years

8. Denison, W. C.: Prickle Cell Epitheliomata of the Ear with Involvement of the Facial Nerve in the Tympanum, Laryngoscope **44**:927-930, 1934.

9. Guttman, J.: Report of a Case of Carcinoma of the Middle Ear, Laryngoscope **30**:727-728, 1920.

previously. Two months before Guttman saw the patient he had vertigo, purulent discharge and paralysis of the sixth and seventh nerves. A mastoidectomy was performed by the attending aurist. When Guttman subsequently examined the patient, there was complete loss of hearing on that side and no response of the labyrinth to the cold caloric test. The reaction of the labyrinth on the opposite side was sluggish. At an exploratory operation, large masses of malignant tumor were removed from the antrum. The pathologist reported this tumor to be squamous cell carcinoma. The patient died six weeks later of purulent meningitis.

Warner [10] has reported the removal of a squamous cell carcinoma from the helix of the left ear of a man of 77. The immediate exciting cause of this condition seemed to be excessive exposure to the sun's rays, to which the patient had not been accustomed. The cartilage of the pinna had kept the carcinoma from invading the deeper tissues, as it so often does.

Fisher,[11] according to Warner,[10] cited the London Hospital Report as saying "that of two hundred thousand cases of all kinds admitted each year, but thirty cases of carcinoma of this organ [the ear] were encountered in a period of ten years' time," or an average of 3 each year. At St. Bartholomew's Hospital 7 cases were encountered in two years. The reports of the Liverpool Eye and Ear Hospital showed treatment given in 2 cases in ten years. At the Mayo Clinic, according to Fisher,[11] there were 27 cases from Dec. 31, 1907, to Oct. 31, 1913. Fisher compiled 22 other cases, from the literature. More cases have been reported in the past twenty-five years than in the preceding twenty-five.

Wolff [12] commented on the infrequency of carcinoma of the external ear. He quoted Haig as saying he was able to collect only 16 cases of tumor of any sort in this situation, while Alexander gathered from the literature 14 cases, in only 8 of which the condition was malignant.

Newhart [13] reported a case of carcinoma of the middle ear in 1917. He reviewed the literature thoroughly. His article is illustrated by six photomicrographs and a photograph of the right side of the head showing the extensive destruction of the external and middle ear. Newhart

10. Warner, F.: Carcinoma of the External Ear, with the Report of a Case, Ann. Otol., Rhin. & Laryng. **26**:950-960, 1917.

11. Fisher, C.: Epithelioma of the External Ear, in Collected Papers by the Staff of St. Mary's Hospital, Mayo Clinic, 1913, Philadelphia, W. B. Saunders Company, 1914, p. 584.

12. Wolff, J.: Die Lehre von der Krebskrankheit von den altestes Zeiten bis zur Gegenwart, Jena, Gustav Fischer, 1928, pt. 2, p. 1212.

13. Newhart, H.: Primary Carcinoma of the Middle Ear: Report of a Case, Laryngoscope **27**:543-555, 1917.

commented on the few reports in the literature of malignant disease of the ear prior to 1917. He said:

> As early as 1886, Kretschmann [14] collected sixteen cases. In 1899, Zeroni [15] reported five cases of carcinoma of the ear, of which, however, three were of the external ear or of the canal. His very exhaustive study of the literature yielded one hundred and twenty-one cases of carcinoma of the ear reported between the years 1804 and 1899. Of these a moderate number were primary middle ear carcinomata.

Newhart said also, "During twenty-four years Bezold [16] saw but four cases of carcinoma, three of the external ear, and one primary in the middle ear, out of 20,000 otological patients."

Nearly all authors have emphasized that carcinoma is rare in persons under 40. However, Alexander [17] has reported the occurrence of this tumor at 17, Leidler [18] at 17, Nager [19] at 19 and Knapp [20] at 20.

Most of the reports of carcinoma of the middle ear have mentioned chronic suppurative otitis media as at least a predisposing cause. However, Danzinger [21] has reported cases in which the condition was not preceded by chronic suppuration but occurred subsequent to acute otitis.

Urbantschitsch [22] has reported cases of epithelioma and sarcoma in which treatment with radium was successful. Most otologists today prefer surgical excision followed by radium and roentgen irradiation, especially if the diagnosis can be made early.

Kiefer [23] has reported successful surgical removal of carcinoma of the tragus, antitragus, concha and external auditory canal without the subsequent use of radium and roentgen rays, although he did not follow his patient long enough to be sure that there would not be a recurrence.

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14. Kretschmann, F.: Ueber Carcinom des Schläfenbeines, Arch. f. Ohrenh. **24**:231-262, 1886-1887.

15. Zeroni: Ueber das Carcinom des Gehörorganes, Arch. f. Ohrenh. **48**:141-190, 1900.

16. Bezold, F., cited by Treitel, L.: Ueber das Carcinom des Ohres, Ztschr. f. Ohrenh. **33**:152, 1898.

17. Alexander, G.: Die Ohrenkrankheiten im Kindesalter, in Pfaundler, M., and Schlossmann, A.: Handbuch der Kinderheilkunde, Leipzig, F. C. W. Vogel, 1912, vol. 6, pp. 1-435.

18. Leidler: Das Mittelohrkarzinom im Lichte moderner Krebsforschung, Arch. f. Ohrenh. **77**:177-188, 1908.

19. Nager, F. R.: Ueber die Bildung von Labyrinthsequester bei Mittelohrkarzinom, Verhandl. d. deutsch. otol. Gesellsch., 1908, pp. 130-135.

20. Knapp, H., cited by Berens, T. P.: A Case of Epithelioma of the Middle Ear, Tr. Am. Laryng., Rhin. & Otol. Soc. **11**:452-456, 1906.

21. Danzinger, E.: Beitrag zur Kenntnis des Felsenbeinkarcinom, Arch. f. Ohrenh. **41**:35, 1896.

22. Urbantschitsch, V.: Ueber einige mit Radium behandelte Ohrenfälle, Arch. f. Ohrenh. **96**:15-21, 1914.

23. Kiefer, H. A.: A Case of Carcinoma of the Pinna and External Auditory Canal, Ann. Otol., Rhin. & Laryng. **20**:71-73, 1911.

On the other hand, Saunders and Callison [24] tried treatment with radium alone for eighteen months for a carcinoma of the auricle, without success, and finally removed half of the auricle to prevent recurrence. They did not say whether or not irradiation was used subsequent to the surgical excision.

REPORT OF CASES

CASE 1.—S. P., a married white American man aged 79, a ranchman, was admitted to the hospital on Jan. 16, 1926, with a diagnosis of carcinoma of each external ear. He was discharged on January 21 as unimproved.

His family history was essentially irrelevant.

His own history included the usual diseases of childhood, with uneventful recovery. His general health had always been good until six months before admission, when he began to lose weight. He had lost 30 pounds (13.6 Kg.).

The illness for which he was admitted had begun as a small pimple in the left ear six months before. This gradually spread. The right ear had become involved six years before in much the same way. The left ear had caused more pain than the right.

The physical examination showed arcus senilis on each side, with about 50 per cent vision on the right. All the teeth had been removed. The throat was normal. The lungs were hyperresonant. The heart was enlarged and the sounds faint. The first sound at the apex was not heard. The aortic second sound was greater than the pulmonic sound. The blood pressure was 210 systolic and 90 diastolic. The abdomen was normal.

The left toes had been amputated after being frozen. There was a large knob on the left tibia from a previous fracture. The third right finger had been removed at the last joint.

There was a hole through the subcutaneous tissues behind the right ear, about ¼ inch (0.6 cm.) deep, from an ulcer, with a necrotic surface. There were extensive induration and redness about the left ear, with extensive ulceration and necrosis. This extended through all the layers of tissue of the auricle. There was some secondary infection, with much pus.

The urinalysis showed no abnormality. The blood count showed 4,050,000 erythrocytes, 8,350 leukocytes and 92 per cent hemoglobin (by the Dare method).

The clinical diagnosis was carcinoma of each external ear. A biopsy was not performed. The patient was examined by several members of the staff in consultation.

He was treated with 2 per cent solution of mercurochrome to lessen the bad odor. Irradiation was not used during his short stay, of five days, in the hospital.

At the request of his relatives and friends he was discharged on January 21 to the Colorado State Soldiers and Sailors Home, Homelake, Colo.

CASE 2.—W. B., a white German married man aged 83, retired, was admitted on Jan. 5, 1932, with a diagnosis of carcinoma of the left ear. He died on January 10.

The physical examination revealed him to be uncooperative. He was irrational and appeared acutely ill. His scalp was normal. His pupils were small and equal. They reacted to light. There was a wide arcus senilis. The right lens was

24. Saunders, T. L., and Callison, J. G.: Carcinoma of the Auricle: Radium Treatment for Eighteen Months with Failure to Cure; Removed with Half of Auricle; Plastic Operation to Cover Defect, Ann. Otol., Rhin. & Laryng. **27**:1060, 1918.

opaque and white. The left lens was less opaque. The right membrana tympani showed numerous perforations. The lobe of the left auricle was partly destroyed by an ulcerated area below the external canal. The nose was normal externally. The teeth had all been extracted. The tongue showed fissures. The pharynx was injected and contained mucopus. It was difficult to see.

The neck was emaciated. There was marked venous pulsation. The chest was hyperresonant. There was slight diminution in resonance in the right axilla. The excursions were somewhat limited on the right. The breath sounds were harsh in the right subclavicular region and in the right axilla. Numerous sticky rales were heard in the upper portion of the right side of the chest posteriorly. The cardiac rate and rhythm were normal. The sounds were faint. The blood pressure was 132 systolic and 50 diastolic.

The abdomen and the genitalia were normal. The extremities were emaciated. The abdominal reflexes were present. The patellar reflexes were absent. There was no Babinski sign. The skin was atrophic, and there were several areas of senile keratosis on the back.

The diagnosis was carcinoma of the left ear and bilateral bronchopneumonia.

The roentgen examination of the chest on January 6 showed the lower half somewhat hazy, with marked increase of peribronchial thickening. The parenchyma of the lungs was otherwise clear. The costophrenic angles were clear. The heart was displaced to the right. There was considerable widening of the aortic arch. The radiologist reported incipient bronchopneumonia over the left base.

The urinalysis showed no abnormality except for a few pus and epithelial cells. The examination of the blood (Dare method) showed 92 per cent hemoglobin. There were 3,970,000 erythrocytes and 7,500 leukocytes on January 6. A Wassermann test on January 8 was negative. A biopsy was not performed.

The patient died in the hospital on January 10 of bronchopneumonia.

During the patient's five days in the hospital he was treated with salicylates, sinapisms, digitos and codeine and given diathermy. He was irrational and had high fever.

Case 3.—H. H., a white German widower aged 66, a commission man, was admitted on Jan. 19, 1932, with a diagnosis of carcinoma of the right external ear, the right side of the face near the ear and the right temporal bone. He was discharged as improved on February 8.

His chief complaint on admission was of the condition of his ear.

His mother had died at 93 of an unknown cause and his father at 50 of an unknown cause. He had 2 brothers and 1 sister living and well. He had 1 brother and 2 sisters dead of unknown cause. There was no family history of cancer, tuberculosis, diabetes or mental disease.

He had been married twice. His first wife had died of pneumonia three years after their marriage. His second wife had died of diabetes three years before his admission to the hospital. He had 3 boys living and well at the ages of 13, 16 and 25. He had 4 children dead. Two had died at 5 or 6 weeks and the others when very young. He did not know the causes of death.

He had lost his appetite, although he had been a hearty eater. He had slept well except when his ear had bothered him. He had smoked a pipe or cigar but not often. He had used coffee and tea in moderation. He had not had any childhood diseases but he had had typhoid fever, malaria, smallpox and pneumonia. He had not been injured.

Three years before admission his right ear had been frozen. Later he had noticed a reddish blue nodule, about the size of a nickel, on the helix. He had neglected the growth, but he had used salves. Two years before admission the tumor had

been removed. Six months later it had been removed again. This time he had received ten or eleven roentgen treatments, and the wound had seemed to heal. Fifteen months before admission he had begun to notice a small nodule. This had gradually become larger. It had also become infected and bled. He had applied poultices to draw the tumor. He was beginning to have some headache. He believed his hearing was normal. He had occasional sore throat. He did not have dyspnea, pain or cough. He had not needed a physic. He had lost 20 pounds (9 Kg.) during the six months before admission.

The physical examination showed him to be fairly well developed; he lay comfortably in bed in no immediate distress but looked rather poorly nourished. His pupils were equal and regular. They reacted to light and in accommodation. The movements of the eyes were normal, and the vision was normal. There was no redness and no discharge.

The right auricle was missing. The left ear did not show any tophi or discharge. The hearing was impaired. There was an ulcerated area about 4 inches (10 cm.) in diameter about the region where the right auricle had been. The floor of this was covered with necrotic tissue and exudate. The margins were indurated and typically carcinomatous. The odor was foul.

The nose was grossly normal. The teeth were in poor condition and the oral hygiene bad. The tonsils were atrophic and the pharynx slightly injected. There was no apparent adenopathy. There was no tracheal deviation or tug and no enlargement of the thyroid. The chest was symmetric, and there was no costovertebral or vertebral tenderness. The percussion note was a little high pitched throughout. The expansions of the chest were equal. The vocal fremitus was slightly reduced. The sounds of breath, voice and whisper were distant but of normal quality. There were no rales. The bases of the lungs were mobile. The pulmonic first sound and the borders of the heart were within normal limits. The cardiac sounds were of normal intensity, quality and duration. There were no murmurs. The aortic second sound was greater than the pulmonic second. The blood pressure was 125 systolic and 65 diastolic. The pulses were equal and regular. There was some arterial thickening.

The abdomen was relaxed and the edge of the liver easily felt. There were no abnormal masses. There were no hernias or dilated veins. There were no tumors or deformities of the extremities. The reflexes of the quadriceps and biceps were active. The genitalia were normal. The prostate was small and normally firm. There were no nodules or areas of softening and no pathologic tenderness.

The clinical diagnosis was carcinoma of the right auricle and the right side of the face.

The neurologic examination, by a consultant, showed no abnormality.

The urine was normal. The blood count showed 5,980,000 erythrocytes, with 83 per cent hemoglobin, and 14,000 leukocytes.

The radiologist reported marked fibrosis in the hili and the bronchial system. There was a small infraclavicular calcification on the right side (old Ghon tubercle). The parenchyma of the lungs and the costophrenic angles were clear. The heart was of normal size. There was marked aortic accentuation. There was no roentgen evidence of invasion by a tumor. There was a large defect of the soft tissue in the right auricular region, possibly involving bone. There was no roentgen evidence of involvement of the right mastoid. The left mastoid was rather small and somewhat hazy.

The otolaryngologist who examined the patient on February 4 found that he could hear a whisper at 1 foot (30 cm.) and the voice at 10 feet (305 cm.).

The lowest fork heard was the 128. The Rinne test showed a ratio of 22 to 16. The Weber test indicated lateralization to the right. There was no spontaneous vertigo, nystagmus, past pointing or falling, and the Romberg sign was not present. Rotary nystagmus was produced by cold irrigation in twenty-two seconds and lasted thirty seconds. The amplitude was good in horizontal and vertical positions. There was normal past pointing with both arms.

The carcinoma was removed, with the patient under nitrogen monoxide anesthesia, on January 20, and the area was cauterized. The tumor was excised approximately 1 inch (2.5 cm.) beyond the margin with the diathermy knife. The remainder of the mass was curetted away and was found to involve the mastoid portion of the temporal bone and a portion of the parietal bone. Both the inner and the outer cells were found diseased and were removed, the lateral sinus and dura overlying the cerebellum being exposed. The granulations were removed from the dura and sent to the laboratory for examination. Simple mastoidectomy was performed, with curettement of the bony external auditory canal. The exposed portions of the muscles of the temple and scalp, the attachment of the sternocleidomastoid muscle and the edges of the skin were cauterized with a hot soldering iron. There was no paralysis of the facial nerve after the operation.

The pathologist made the following report:

"The specimen consists of tissue from the auricle and the dura. The tissue from the auricle measures 3 by 2.5 by 1 cm. It is ovoid and slightly lobulated. The surface is pale yellowish pink and resembles normal skin. At one part the tissue is firm. When cut, this area has a white opaque center surrounded by translucent material with yellow spots in it.

"The other piece of tissue measures 0.5 by 0.4 by 0.2 cm. It is red and of a granular appearance.

"The microscopic sections showed a layer of stratified squamous epithelium overlying fibrous connective tissue, cartilage and fat. The epithelium was thin and had shallow interpapillary downward growths. Here and there sebaceous glands were seen. In one area the epithelium projected deep into the subcutaneous tissue, forming cords and groups of atypical epithelial cells. These cells were round and polygonal but varied greatly in size. The nuclei varied greatly in size and shape and were round to oval, with prominent nucleoli and mitotic figures. The fibrous tissue was proliferative and packed with plasma cells and lymphocytes. The cartilage and adipose tissue were normal in appearance.

"The section of the dura showed a large amount of proliferating fibrous tissue, containing solid cords and groups of the type of atypical cells described.

"The pathologic diagnosis was squamous cell carcinoma of the auricle with extension to the dura."

The patient was readmitted to the hospital on April 12 for reexamination. The right auricle was eroded, and in its place was an ulcerated, circular erosion 5 cm. in diameter and several centimeters deep, the floor presenting a necrotic foul dirty typically carcinomatous mass. This condition prevailed in spite of irradiation, undertaken in the outpatient department after the operation on January 20. The patient complained of more headache, probably from the involvement of the dura.

On April 13, with the patient under anesthesia induced by amytal, avertin with amylene hydrate and nitrogen monoxide, the granulations were curetted away from the exposed dura and overlying bone. The edges of the bone were found to be necrotic and were removed with the rongeur. The entire wound was cauterized with the electric cautery. A petrolatum gauze dressing was applied.

The patient was discharged to the outpatient department as unimproved on April 15 for subsequent irradiation.

The two photographs (fig. 2) show the condition soon after admission to the hospital and the more advanced involvement shortly before discharge.

CASE 4.—A. G., a white widow aged 63, whose occupation was housework, was admitted on March 13, 1935, with a diagnosis of carcinoma of the left ear. She was discharged on leave on May 24.

Her chief complaint was of a purulent ulceration anterior to the left ear.

Her mother had died of an unknown cause at 41. Her father had died of Bright's

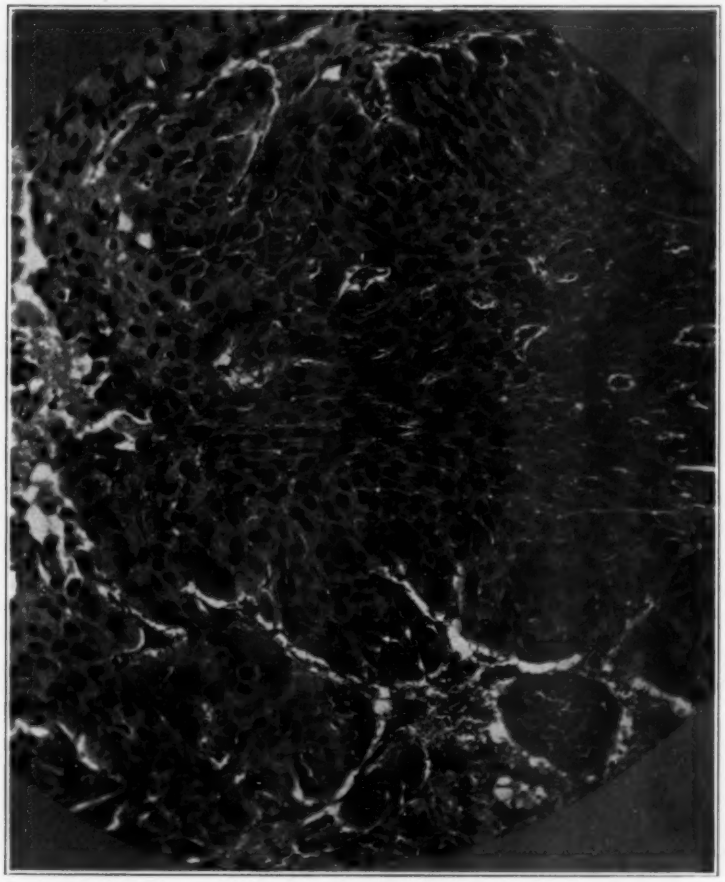

Fig. 1 (case 3).—Photomicrograph of squamous cell carcinoma of the auricle with extension to the dura.

disease at 62. She had 2 brothers and 2 sisters living and well. One sister had died of an unknown cause at 65.

There was no family history of cancer, tuberculosis, asthma, hay fever or nervous or mental disease.

The patient had not used tobacco, alcohol or drugs. Her marital history was uneventful except that she had lost 1 child from diarrhea and 1 from diphtheria during their early childhood. Her husband had died at 70 of cardiac disease.

She had had mumps in childhood but no other diseases, and she stated that she had had no venereal disease.

About thirteen years previously she had noticed a raised roughened area the size of a pinhead anterior to the ear. This did not cause pain and did not grow. Two years before admission it had enlarged to about the size of a dime. Her family physician had given her a salve to use. After she had used the salve for a short time, the area had become eroded, and there had been a purulent discharge ever since.

The physical examination showed her to be well developed and poorly nourished; she lay quietly in bed, apparently in no acute distress. The scalp was clean and without scars and was covered by grayish brown hair. There were no exostoses of the skull. The right ear was normal. The left was indurated and ulcerated, with purulent discharge. The movements of the eyes were normal. The pupils were equal and regular. They reacted to light and in accommodation bilaterally. The external part of the nose was normal. The mouth was edentulous, and the

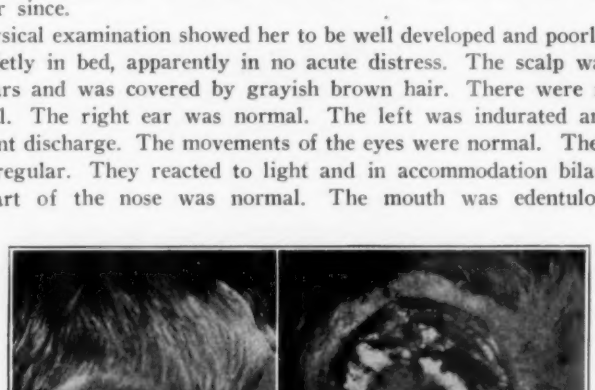

Fig. 2 (case 3).—Photographs taken on Jan. 5 (*A*) and Jan. 29 (*B*), 1932, showing the rapid spread of the carcinoma in twenty-four days. The patient died late in 1932, probably of carcinoma of the cerebellum.

mucous membranes were pale. The tonsils were atrophic. The pillars were slightly injected.

The thyroid gland was not enlarged. The signal node was palpable. There was no lymphadenopathy on the right. The pulmonary fields were resonant throughout. There were no rales and no abnormal breath sounds. There was no dulness. No enlargement of the heart could be detected by percussion. The cardiac rate was 90. The rhythm was regular. A systolic murmur was heard over the mitral area. The aortic second sound was greater than the pulmonic second. The blood pressure was 148 systolic and 46 diastolic.

The abdomen was distended and pendulous. There was an absence of fluid, masses and tenderness. The liver and spleen were not palpable. The extremities were apparently normal. The reflexes were normal.

The clinical diagnosis was carcinoma of the left ear.

The reports from the laboratory showed the urine to be normal except for a few pus and epithelial cells, calcium oxalate crystals and bacteria at four examinations from March 14 to May 13. The hemoglobin content was 12.2 Gm. per hundred cubic centimeters by the Sahli test. The erythrocyte count

ranged during the aforementioned period from 3,860,000 to 3,220,000 and the leukocyte count from 4,100 to 6,200. The Wassermann and the Eagle test were negative.

On March 15, with the patient under anesthesia induced by avertin with amylene hydrate and gas, the entire area of the carcinoma was coagulated. The sheath of the parotid gland was entered and thoroughly cauterized. A dry dressing was applied. No paralysis of the facial nerve followed the operation.

The pathologist reported that the gross specimen from the left ear measured 1.4 by 0.7 by 0.4 cm. The external surface was apparently roughened skin discolored brown and sharply demarcated from a small portion of pink epithelium. The surface of the section was similar, brown connective tissue being abruptly demarcated from pink connective tissue.

Microscopically the specimen consisted of epithelium and subepithelial connective tissue. The squamous epithelium was partly cornified and appeared fairly normal over half of the section. The normal epithelium shaded into a blue-staining, necrotic-appearing surface. The connective tissue was hemorrhagic, edematous and infiltrated with polymorphonuclears, lymphocytes and monocytes.

The pathologic diagnosis was acute inflammation and necrosis of skin and subcutaneous tissue without evidence of malignancy.

The pathologist examined also a specimen of 500 cc. of light yellow ascitic fluid submitted by the consulting internist, who suspected the presence of malignant growth in the abdomen. There was a finely granular flocculation in the fluid.

Microscopically there were many cells. Some appeared to be crenated red blood cells. The rest included several types of cells. Some were fairly large, with pinkish-stained reticulated cytoplasm and deeply blue-stained nuclei. Some of these had what appeared to be pinkish-stained vacuoles in the cytoplasm. Other cells were smaller, with nuclei almost filling them. Still others had polymorphous nuclei. Some cells contained nuclei. There was an occasional group of cells. One such group contained seven concentrically arranged cells with one cell more deeply embedded and in the center. Throughout the section there was collagenous material.

The pathologist said the sediment of the ascitic fluid showed no evidence of malignancy.

The ear was dressed daily with gauze wet with solution of merthiolate, at first of 1 to 1,000 strength and gradually weakened to 1 to 5,000.

The ulcer healed slowly but not entirely. There were less discharge and less foul odor, but the healing was not complete.

Radium had been used elsewhere before the patient was admitted to Colorado General Hospital. Radium and roentgen rays were not used while she was in the hospital.

She was discharged on leave as unimproved on May 24. She died on Feb. 27, 1936.

Clinically this patient had carcinoma, but the pathologic report did not confirm this. The specimen taken for biopsy may not have included the malignant area, or the irradiation in another hospital before she came to the Colorado General Hospital may have destroyed the malignant calls, temporarily at least.

CASE 5.—C. D. H., a white boy aged 4 years, was admitted on Feb. 3, 1936, with a clinical diagnosis of malignant disease of the right middle ear. The patient was discharged on May 2 as unimproved and died in another hospital on June 12.

The chief complaint was of a growth in the right ear of one year's duration.

In December 1934 the child had had pneumonia complicated by acute suppurative otitis media. He had apparently made a good recovery from both. In February 1935 his mother had noticed a small red growth protruding from the right ear. This was treated with ultraviolet rays for about two months. The growth was removed in June. Subsequent to the removal there were discharge and frequent frank hemorrhages.

In spite of all therapy the tumor continued to grow; it was removed a second time in August. After August the growth was repeatedly clipped off with a snare. In November a nodule appeared below the right ear. The patient's condi-

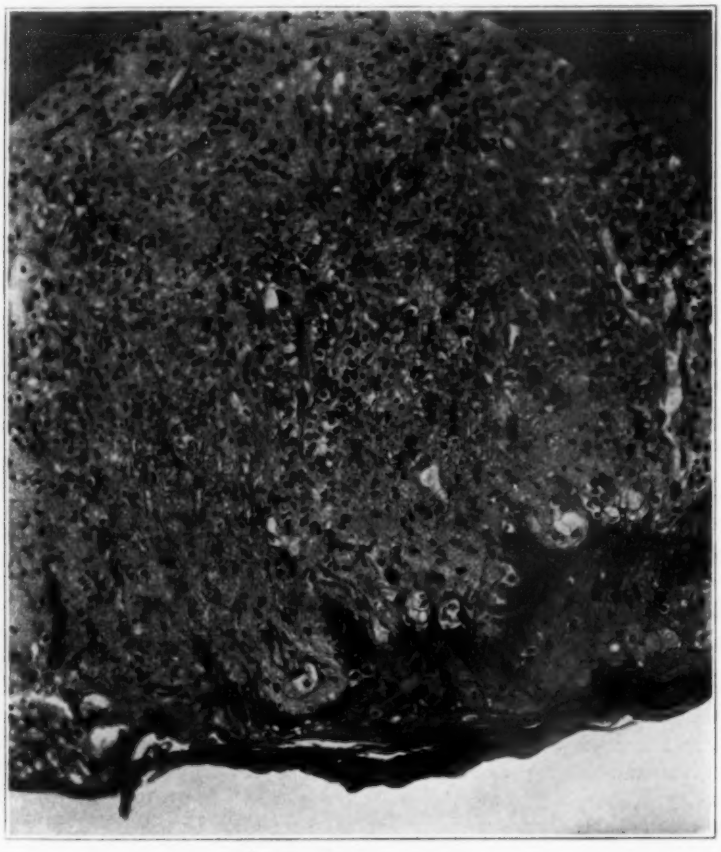

Fig. 3 (case 4).—Photomicrograph showing acute inflammation and necrosis of skin and subcutaneous tissue without evidence of malignancy.

tion gradually became worse, and he lost weight. A mastoid operation was performed late in November, but the tumor kept on growing. It did not, however, produce pain.

The father was 41 and well. The mother was 36 and well. A sister of 3 was well.

There was no history of cardiac disease, diabetes, tuberculosis or nervous disorders. The paternal grandmother had died of carcinoma of the stomach. The maternal grandfather had died of Bright's disease.

The physical examination showed a small, markedly undernourished white boy, anemic, apathetic and apparently sick. The skull was symmetric and without exostoses. There were no scars or ulcers of the scalp. The hair was brown. The face was paralyzed on the right. The pupils were regular, but the right was smaller than the left. Both reacted to light and in accommodation. There was no nystagmus and no exophthalmos. There was slight ptosis of the right upper eyelid. The external rectus muscle on the right was paralyzed. The movements of the left eye were normal. The left ear was normal. A red tumor in the right external auditory canal exuded serous material. The whole region posterior and inferior to the pinna was swollen and somewhat tender. The incisional cicatrix in the posterior inferior mastoid was draining a small amount of seropurulent matter through one opening about 2.5 by 0.5 cm. and another just below the pinna 1.5 by 0.75 cm. The tumor was firm and smooth. It extended anteriorly and made the face asymmetric, but was not inflammatory. The nose was normal externally, and the teeth were normal. The tongue was coated. It deviated to the right but could be moved to either side. The tonsils were slightly injected and so was the pharynx. There was some paralysis, with relaxation of the soft palate, on the right. The reflexes were present. Both vocal cords moved normally.

The neck was slightly swollen on the right. The thyroid was not palpated. There were no pulsations in the neck, but it seemed stiff. There was poor mobility. The examiner believed there was some weakness of the right accessory nerve. The thorax was thin and poorly developed. The expansions were equal. There were no pulsations or thrills. The lungs were resonant throughout. There were no rales. The breath sounds were normal. There was normal transmission of the whispered voice. The cardiac sounds were normal. There were no murmurs. The sounds were of good quality and rhythm. The heart was not enlarged to percussion. The point of maximal impulse was at the fifth interspace. There were no thrills or pulsations.

There was no tenderness of the abdomen and no masses or rigidity. The genitalia were normal. The inguinal rings were not enlarged. The extremities were normal except for emaciation. The reflexes were physiologic and equal. The back was normal. The lymphatic glands were palpable because of emaciation.

The clinical diagnosis was tumor of the right middle ear, possibly malignant, with involvement of the sixth, the seventh, the eighth, the ninth and the twelfth cranial nerve.

The report from the laboratory showed the urine to be normal. The blood count showed 5,080,000 erythrocytes, with 11 Gm. of hemoglobin per hundred cubic centimeters by the Sahli test, and 9,200 leukocytes, with 60 per cent polymorphonuclears and 33 per cent lymphocytes. Serologic tests were not made.

On Feb. 5, 1936, with the patient under anesthesia induced by ethyl chloride, a biopsy specimen was taken from the tumor.

The pathologist reported that the specimen consisted of three small pieces of rough coarse tissue, partially fixed. Each had a rough and a smooth surface.

On microscopic examination the sections were seen to contain a thin squamous epithelium, which dipped deeply into the stroma. There was a break in the continuity of this, which was covered by granulation tissue consisting chiefly of polymorphonuclear leukocytes, monocytes and fibrin. The stroma contained many foamy cells, some polymorphonuclear leukocytes and many large pale cells which occasionally contained two and even three nuclei. No actual mitosis was seen.

The pathologic diagnosis was myxosarcoma of the right ear.

High voltage roentgen therapy was given on February 5 and repeated on February 6 and 7. There was a severe hemorrhage from the right ear on February 8, which was controlled by packing. The patient was stuporous most of the time.

The patient was discharged as unimproved on May 2 and died in another hospital on June 12.

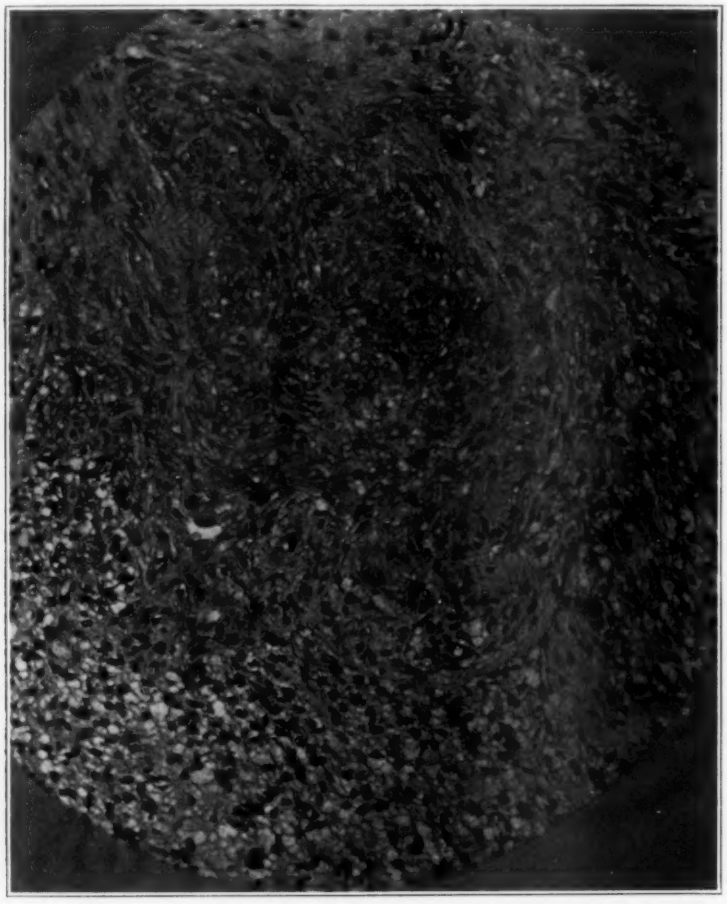

Fig. 4 (case 5).—Photomicrograph of myxosarcoma of the right external ear.

CASE 6.—J. L., a white widower aged 66, a farmer, was admitted on May 12, 1936, with a clinical diagnosis of carcinoma of the left ear. He was discharged on leave four times before his final discharge as worse on October 2.

His chief complaint was of chronic discharge from the left ear for seventeen or eighteen years and more recently of an ulcer of the concha. His ear had sometimes stopped discharging, until two years previously, when he had had his teeth extracted. Since then it had discharged constantly. He had had some rheumatism, particularly of the right hip.

His father had died at 70 of an unknown cause and his mother at 70 from the late effects of the menopause. He had lost 1 brother after an operation. He had 4 brothers living and well. There was no family history of tuberculosis, nephritis, diabetes, asthma, cancer or cardiac disease.

He had not used alcohol, but he had used some tobacco. For three or four weeks before admission to the hospital he had taken four acetylsalicylic acid tablets daily.

His wife had died of cancer of the uterus at 36. One son aged 40 and 1 daughter aged 30 were living and well. Two children had died at birth. He believed these deaths were caused by miscarriage, possibly self induced.

He had had measles, mumps and pertussis before he was 30 but no other diseases except rheumatism. He had undergone tonsillectomy and an operation for left inguinal hernia twenty-one years before admission. He had fallen through a manhole and broken his left leg eight years before admission. He did not believe he had lost any weight. He had not suffered from acute rheumatism, but the chronic rheumatism had been worse for eighteen months.

The physical examination showed him to be well developed, well nourished and in no acute distress. His scalp was fairly clean. His forehead had a scaly area over the right eye. His pupils were equal and regular. They reacted slightly to light. The movements of the eyes were normal. The right external ear was normal. The left ear had a lesion in the lower part externally, just below and behind the external canal. There was a shallow, punched-out ulcer with a bleeding surface anterior to which was a whitish slightly elevated area. The nose was normal externally. All his natural teeth had been removed. His tongue protruded in the midline without tremor.

There was some cervical adenopathy on the left. The thyroid was not enlarged. The expansions of the chest were equal bilaterally. There was some retraction of the soft tissue over the apex of the right lung. The cardiac sounds were faint. The first sound at the apex was split. There were no murmurs. The rhythm was regular. The pulmonic first sound was heard best at the fourth interspace, inside the nipple line. No enlargement of the heart could be detected by percussion. The blood pressure was 110 systolic and 70 diastolic. The lungs were hyperresonant to percussion anteriorly. The breath sounds were faint and rather blowing. There were no rales. The vocal fremitus was not increased. The tactile fremitus was normal. The lungs did not expand posteriorly with any satisfaction. The breath sounds were faint, and the expiration was increased in duration.

There were no masses in the abdomen. There were some tenderness and a slight dulness to percussion in the right lower quadrant. There was a scar in the left lower quadrant above and parallel to Poupart's ligament. The right inguinal ring was relaxed. However, no definite cough impulse was elicited. The reflexes were difficult to obtain because of atrophy and stiffness of the joints from rheumatism but seemed to be normal. The abdominal reflexes were present. The rectum was normal.

All joints of the hands and arms were stiff and painful on motion. There was some limitation of motion of the hands, which were deformed. The right was the worse in adduction. The finger nails looked atrophic, and there was wasting of the muscles. The feet appeared swollen, and their motion was limited. Motion caused pain. The knees and hips were limited in motion and painful. There was atrophy of the muscles of the upper part of the legs. There was a scar on the left thigh laterally. There were scars from former bedsores on the back and buttocks. The skin was hyperkeratitic and scaly in patches not well defined.

The clinical diagnosis was carcinoma of the left ear with atrophic arthritis.

A specimen was taken for biopsy on May 14, with the patient under local anesthesia induced by 1 per cent solution of procaine hydrochloride. A small piece of the lesion was excised and sent to the pathologic laboratory for examination and diagnosis.

The pathologist said the specimen consisted of a sliver of tissue 0.8 cm. in length; this was fixed with a solution of formaldehyde.

The microscopic examination revealed squamous epithelium dipping beneath the epithelium. There were a few mitotic figures. There were in other areas many polymorphonuclear leukocytes in fibrin. The tissue was of a fairly well differentiated type.

The pathologic diagnosis was suppurative inflammation of tissue from the left ear. There was no evidence of malignancy. The pathologist suggested that a larger piece be removed and another biopsy performed.

A second specimen was accordingly taken for biopsy on May 27, with the patient under local anesthesia induced by freezing with ethyl chloride. A larger piece of tissue was removed with a cold sharp scalpel and the bleeding checked by coagulation. The pathologist said the specimen consisted of one piece of tissue 3 by 1 cm. and two smaller pieces of tissue.

The microscopic examination showed the epithelium was squamous and dipped deeply into the stroma. There were nests of epithelial cells in the stroma. Mitotic figures were not common. A piece of cartilage had some calcification. Most of the areas showed the characteristics of basal cell carcinoma, but in some there were areas resembling squamous cell carcinoma.

The pathologic diagnosis was carcinoma of the ear, predominantly of the basal cell type.

On May 11 the urinalysis showed a trace of acetone, a 2 plus reaction for hyalin and finely granular casts. Pus cells were present (1 plus). There were a few squamous epithelial cells and phosphate crystals. The amorphous débris was rated 2 plus.

On May 12 there were 4,870,000 erythrocytes and 10,280 leukocytes, with 80 per cent polymorphonuclears, 17 per cent lymphocytes, 2 per cent endothelial cells and 1 per cent eosinophils. The hemoglobin content was 12.6 Gm. per hundred cubic centimeters by the Sahli test.

On May 16 the radiologist reported extensive atrophic arthritis involving the knees, wrists and hands, combined with marked bony atrophy of all visualized bones and subluxations and luxations in the phalangeal joints of the hands and feet and the metacarpophalangeal and metatarsophalangeal joints. There were hypertrophic arthritis, combined with scoliosis in the lumbar spine, and marked sclerosis of the abdominal aorta.

The patient was examined from time to time by different members of the staff in consultation. Irradiation seemed to be the best method of treatment and was employed as follows:

Date	Area, Mm.	Distance from Tube, Cm.	Kilovolts	Milliamperes	Filter	Time, Min.
6/ 9	15 by 15	50	180	30	5 mm. Cu, 2 mm. Al	10
6/11	15 by 15	32	110	5	0.5 mm. Al	4
6/12	15 by 15	30	110	5	0.5 mm. Al	4
6/16	15 by 15	32	110	5	0.5 mm. Al	4
8/11	15 by 15	35	110	5	0.5 mm. Al	5
8/14	15 by 15	32	110	5	0.5 mm. Al	5

After the last treatment the patient was asked to return in four weeks.

On September 23, 24 and 25, 1,200 milligram hours of radium irradiation was given to the epithelioma of the left ear. For this five needles containing 5 mg. each, with 0.5 mm. of platinum filtration were used at a distance of 1 cm. for forty-eight hours.

When the patient was finally discharged, on October 2, his condition was poor, although the ulcer had healed.

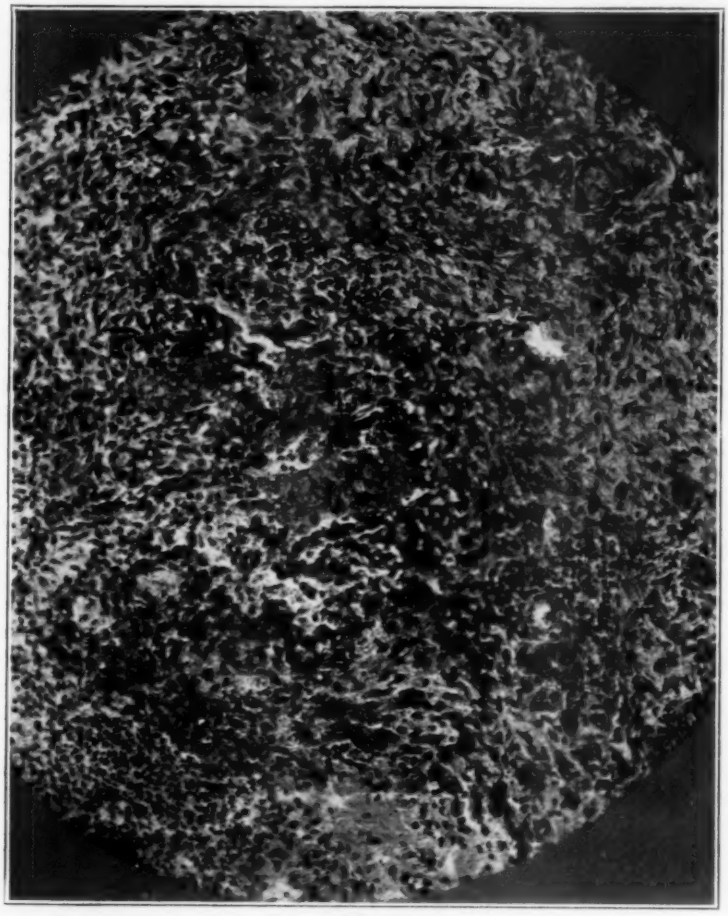

Fig. 5 (case 6).—Photomicrograph of basal cell carcinoma of the ear.

CASE 7.—T. P. G., a white married man aged 67, a miner, was admitted on July 10, 1936, with a clinical diagnosis of carcinoma of the left ear. He was discharged as improved on August 19.

His chief complaint was of cancer of the left external ear.

His father had died of old age at 80; his mother, of old age at 75. He had 1 brother and 3 sisters dead and 2 brothers and 4 sisters living and well. There was no family history of tuberculous, cardiac, renal, metabolic, allergic, mental or malignant disease.

He had seldom drunk coffee, but he had used weak tea daily. He had not used tobacco, alcohol or drugs. He had taken some liquid medicine for the heart for one year before admission to the hospital.

He had had measles, scarlet fever and influenza, but he had not had any operations. He had sustained a fracture to the patella at the age of 40. He had not had any other diseases.

Six months before admission he had noticed itching of his left ear. He had noticed an ulcerated lesion which bled slightly. He applied salve to this and had it cauterized from ten to twenty times. The lesion continued to grow over the external ear, and he was sent to the hospital for radium treatment.

The physical examination showed him to be of short stature and of medium frame. He was up and moved around the ward without obvious difficulty. The skull was normal. There were no exostoses. He had thin gray hair. The pupils were small, regular and equal. They reacted to light and in accommodation. The movements of the eyes were free and equal. There was no nystagmus. The conjunctivas were normal, but the scleral vessels were dilated.

The bridge of the nose was sunken from a former fracture, but there was no obstruction to breathing and no discharge. The right ear was normal. The left showed a purplish discoloration of the upper half of the pinna. The skin was atrophic and hard. The auricle was exquisitely tender. There were several small areas of crusting with old blood.

The mouth was foul. There were many carious teeth, with pyorrhea and marked gingivitis. The tonsils were small and slightly injected. The pillars and pharynx also were injected.

There was marked keratosis of the skin of the neck and face. There was a small sebaceous cyst at the right outer canthus. There was slight anterior cervical adenopathy. The glands were firm and tender on the left side. The thyroid gland was palpable but not enlarged.

The chest showed the asthenic habitus. The lungs were resonant except over the hili posteriorly. The breath sounds were slightly bronchovesicular throughout.

The heart was not enlarged to percussion. The pulmonic first sound was heard at the fifth intercostal space 8.5 cm. to the left of the midline. The sounds were distant and regular. There were no murmurs. The blood pressure was 120 systolic and 80 diastolic. There were marked peripheral tortuosity and arteriosclerosis of the vessels.

The abdomen was soft. There were no masses and no tenderness. The liver, spleen and kidneys were not palpable. The inguinal rings were relaxed, more markedly on the right. The patient was wearing a bilateral truss.

The genitalia were normal externally. There were no scars and there was no discharge.

The extremities did not show any weakness, anesthesia or hyperesthesia. The deep and superficial reflexes were present and equal.

The clinical diagnosis was carcinoma of the left auricle.

On July 10 the report from the laboratory showed the urine to be normal. There were 4,360,000 erythrocytes and 7,050 leukocytes, with 72 per cent polymorphonuclears, 23 per cent lymphocytes and 5 per cent endothelial cells. The Wassermann and the Eagle test of the blood were negative. The hemoglobin content was 11.4 Gm. per hundred cubic centimeters by the Sahli test.

On July 17, with the patient under general anesthesia induced by ethyl chloride and ether, the skin on the posterior aspect of the left ear was dissected free from the body of the pinna. The upper two thirds of the auricle was excised. An attempt was made to reconstruct the ear by folding the posterior flap over the

remaining portion of the pinna. This was sutured in place with interrupted dermal sutures.

The pathologist reported that the specimen measured 4 by 3.5 by 1 cm. It was roughly crescent shaped. The free surface was bluish white and blood tinged, and in many places, where erosion had occurred, the tissue was macerated. The under side was reddish yellow, with a bit of fat adherent to the cartilaginous surface. The consistency was that of cartilage.

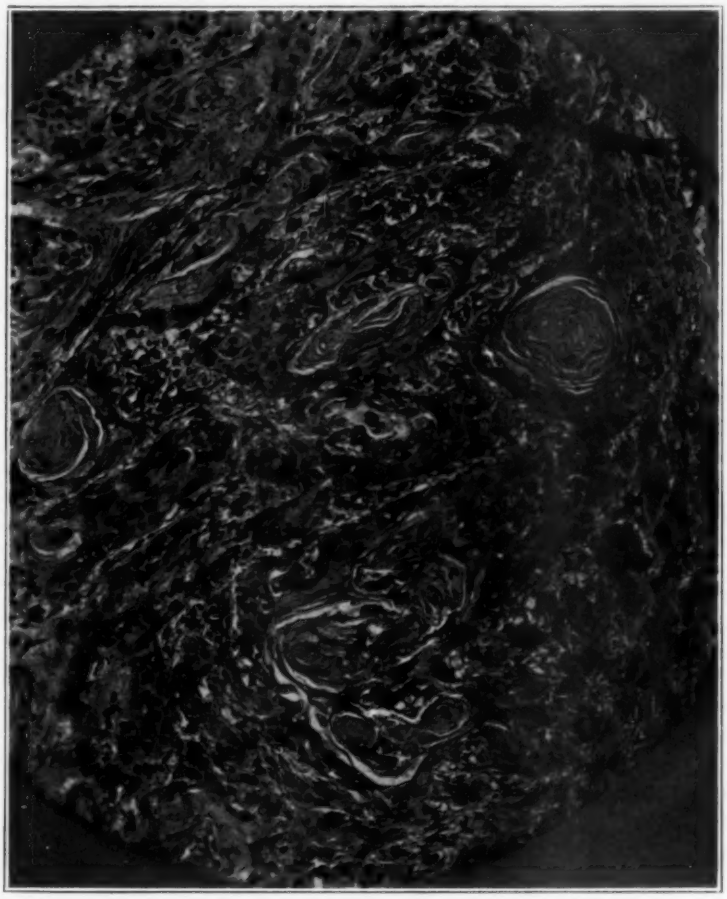

Fig. 6 (case 7).—Photomicrograph of squamous cell carcinoma of the left auricle.

The microscopic section consisted of epithelium, connective tissue and cartilage. In places the stratified squamous epithelium was perfectly regular. In others there was invasion of the prickle cells into the subcutaneous tissue, with pearl formation. In places there was invasion into the cartilaginous cells in stages of mitosis.

The pathologic diagnosis was squamous cell carcinoma of the left auricle.

Late in July a specimen of tissue was removed from the dorsum of the right hand with the patient under local anesthesia. This was disk shaped and measured

1 by 0.4 cm. It was thick at the center. It had a pinkish gray warty surface tinged with blood. The under surface was flat.

Microscopically, at the edges of the section the epidermis appeared normal. In other places the cells which lined the papillae of the corium were beginning to break rank. In the middle the epidermis was ulcerated and replaced by an acute polycytic reaction and many round cells from the corium. The corium everywhere was moderately infiltrated with mononuclears, chiefly small lymphocytes. The keratinized layer of epidermis was markedly heaped up in a few fields.

The pathologic diagnosis was chronic ulcer with keratosis and epithelial hyperplasia.

On July 29 several epitheliomas were removed from the face and neck by electrodesiccation, with the patient under local infiltration anesthesia induced by 2 per cent solution of procaine hydrochloride. Methylrosaniline was applied.

On September 18 a carcinoma about the size of a dime was removed from the right supraorbital ridge with a curet. The edges were sharply defined, and there was no ulceration. The lesion was very tender. The surrounding tissue also was removed with the curet down to normal bone. The area was desiccated down to the bone and a dry crust left.

Irradiation was used for all the lesions. The patient improved under treatment and was discharged as improved on September 25.

CASE 8.—J. D. S., a white widower aged 80, retired, was admitted on Aug. 29, 1937, and discharged on September 13. The diagnosis on admission was carcinoma of the left ear and the nose.

The chief complaints were of a growth on the bridge of the nose and the loss of his left ear.

His father had died at 69 of pneumonia. His mother had died at 35 during childbirth. Two brothers were living and well; 2 died in infancy; 1 of pneumonia in later life and 1 of a stroke. One sister was living and well at 86; another had died of a stroke at 75. His father had had cancer of the nose. There was no family history of tuberculosis or diabetes.

The patient had worked as a farmer, a stone mason and a plasterer. He had drunk one cup of coffee each day and one cup of tea. He had smoked a pipe, but he had not used alcohol.

He had married in 1882. He had 4 children living and well by his first wife, and 1 child had died in infancy. His first wife had died of typhoid fever. He had remarried in 1900 but obtained a divorce seven years later.

He had had chickenpox, whooping cough, measles and smallpox. He had not had diphtheria, scarlet fever or typhoid fever. He had undergone prostatectomy in 1925.

About five years before admission he had noticed a tumor in his left ear. He had gone to a physician who applied paste. About three years before admission a mass had started to grow on the nose. One year before admission some radium had been applied, and considerable destruction had resulted. The tumor had disappeared, but he had had another mass on the bridge of the nose. The tumors of the left ear and the nose had been treated with the electrocautery in addition to radium.

He had suffered from frequent colds and pains in the head. He had had no cardiorespiratory pain and little cough. He had a good appetite and no indigestion but some constipation. He seldom had nocturia.

The physical examination showed him to be well developed but poorly nourished, with a badly disfigured ear and nose. His skin was dry, coarse and warm. There were no exostoses of the skull. The scalp was clean. The hair was thin and gray. The pupils were unequal, the right being the larger, but both were small. Neither reacted to light readily. There was an arcus senilis. The right sclera and conjunctiva were inflamed. The movements of the eyes were normal.

The right ear was normal. The left pinna was gone, only the open external auditory canal remaining. There was a tumor extending into the canal from the anterior wall. The left ear was deaf. The tip of the nose was gone and the septum and turbinates vividly exposed. There was a foul-smelling, dirty-looking mass, about the size of a walnut, on the left side of the bridge of the nose. The patient had a slight cold. All his teeth had been removed. His gums were normal. His tongue was clean and protruded in the midline. His tonsils were small. The anterior pillars were slightly inflamed. The thyroid was not palpable. The trachea was in the midline.

The lungs were resonant throughout. The breath sounds were loud but normal. There were rales in the left posterior base. The vocal fremitus was normal. No enlargement of the heart could be detected by percussion. The pulse rate was 78 and regular. The sounds were distant and faint. There were no murmurs. The aortic second sound equaled the pulmonic second. The blood pressure was 118 systolic and 68 diastolic.

The abdomen showed a cicatrix in the midline below the umbilicus. The abdomen was soft. Considerable gas was palpable in the colon. There was no pain or tenderness. The left inguinal ring was relaxed. The genitalia were not examined.

There was marked sclerosis of the vessels of the extremities. Both fifth fingers were deformed. There was marked flexion, with stiffness. The toes were deformed, and the patient had corns. The reflexes were all sluggish but not pathologic. There was no enlargement of the lymphatic glands.

The clinical diagnosis was carcinoma of the left ear and the nose, probably of the basal cell type.

The report from the laboratory showed the urine to be normal except for 2 pus cells, 1 squamous epithelial cell and 1 mucous shred per high power field. The blood count showed 3,880,000 erythrocytes and 12,300 leukocytes with 85 per cent polymorphonuclears, 14 per cent lymphocytes and 1 per cent endothelial cells. The hemoglobin content was 11.8 Gm. per hundred cubic centimeters by the Sahli test.

On September 2, with the patient under anesthesia induced by avertin with amylene hydrate and procaine hydrochloride, the partial stump of the nose, in which a tumor of approximately 3 cm. had recurred, was removed in toto by means of the electrocoagulating knife. This involved the right inner canthus, but care was taken to avoid severing the tear duct on this side. In a similar manner recurrent carcinoma in the left external auditory canal was removed. The pinna had previously been removed.

The pathologist said the gross specimen consisted of two bits of tissue, measuring 2 by 2 by 1 cm. and 1.5 by 0.8 by 0.8 cm. A grayish green fungating friable surface with a firm pinkish gray base was seen.

Microscopically the stratified squamous epithelium was seen to be greatly thickened and desquamating. Its papillae were greatly hypertrophied and dipped down into a scanty pink connective tissue stroma sprinkled with round cells. The epithelium was denuded for a stretch, giving way to masses and balls of

amorphous pink material containing nests of polymorphonuclears. At the lower edge of this material were wild-growing sheets and cords of cells, with large oval dark and light blue vesicular nuclei and pink cytoplasm. These had invaded the scant stroma, forming numerous pearls. Many mitotic figures were present (graded by Queen as type 1 and by Mulligan as type 3).

The pathologic diagnosis was squamous cell carcinoma of the left ear and the nose.

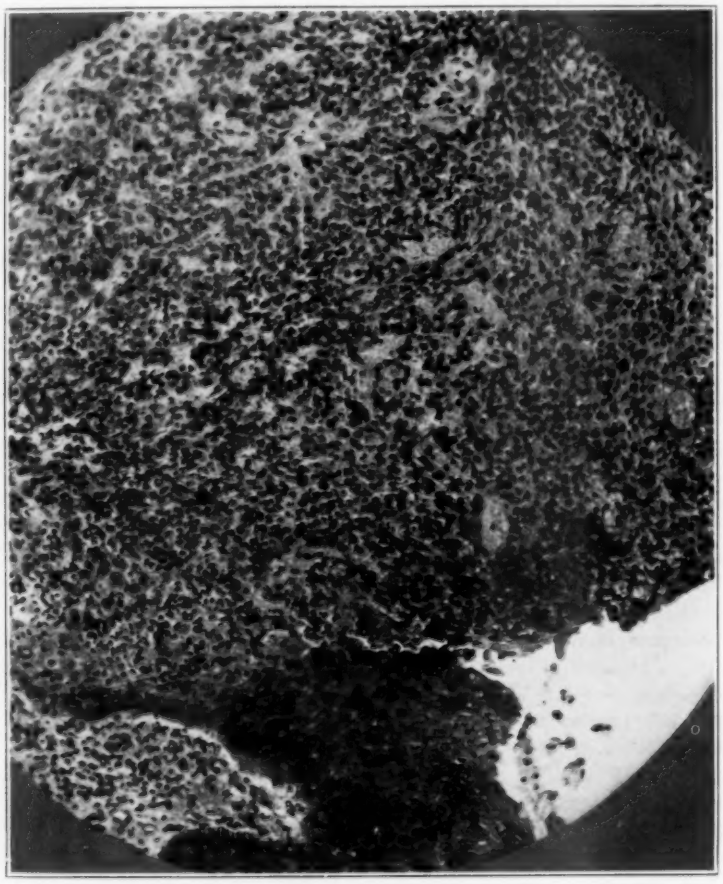

Fig. 7 (case 8).—Photomicrograph of squamous cell carcinoma of the left ear.

The patient's course after operation was rather uneventful. He was discharged on September 17 to a convalescent home as improved.

Results.—The follow-up investigation shows that 3 of the patients are living at the time of this report, but they are not well. Two are known to be dead. The social service department has not been able to locate the other 3.

SUMMARY AND CONCLUSIONS

A series of cases of malignant disease of the ear, including 3 of proved squamous cell carcinoma, 1 of basal cell carcinoma, 1 in which the biopsy specimen failed to include the malignant area, 1 of myxosarcoma and 2 of clinical carcinoma in which biopsy was not performed, is reported.

Carcinoma of the auricle involving the helix only if seen and diagnosed early offers an excellent opportunity for cure. In some cases either the diagnosis was not made early, or the patient refused surgical removal. On the other hand, carcinoma of the external auditory canal and the middle ear has usually invaded the temporal bone so far that complete removal is impossible by the time the correct diagnosis has been made. The literature is not voluminous, as large clinics have had relatively few cases during any ten years. If physicians expect to reduce the death rate from carcinoma—and they should—they must make early diagnoses.

Nearly all the patients when first examined by the family physician received a prescription for a salve. Later they had one or more operations. By the time they were admitted to the Colorado General Hospital they had advanced malignant disease. The high mortality is regrettable, but it cannot be avoided when only patients in late stages of the disease are received.

Dr. William C. Black, of the department of pathology, prepared the histologic specimens, and Mr. Glenn E. Mills, the photographer at the medical school, made the gross photographs and photomicrographs.

Physicians' Building.

ABSTRACT OF DISCUSSION

Dr. H. P. Mosher, Boston: Any one who wants a thorough and up-to-date summary of the literature dealing with this rather rare condition will find it in Dr. Spencer's paper. His own 8 cases are recorded with the greatest care. He did not have a chance with his patients, as they came to him too late. There have been cases of this type in which cure has been obtained. L. A. Schall has developed his own operative technic for dealing with disease of this type. My feeling is that the condition is more common than the literature summarized by Dr. Spencer would lead one to believe.

I fully agree with Dr. Spencer as to his main points, namely, that the condition is relatively rare and that operation followed by the use of radium and roentgen rays is the procedure of choice.

The technic used in this disease will be modified, since the use of roentgen rays and radium is coming along so rapidly. I feel also that the markedly mutilating operations which all otologists used to do and of which they were so proud will be less and less necessary as times goes on.

Dr. Gordon B. New, Rochester, Minn.: Dr. Spencer, in his carefully prepared paper, has given an excellent review of the literature and a careful report of the cases that he has observed. In my experience with this condition, particularly

when it affects the external ear, the diagnosis has been delayed in many cases because the disease was mistaken for some other condition. That is, I have seen low grade malignant lesions mistaken for furunculosis and treated as such. Some of them have been drained, and some have been given roentgen treatment for inflammation, and then later a biopsy has been made and the true nature of the condition has been discovered.

It seems to me that if specimens are removed for biopsy in all cases in which there is doubt of the true condition, particularly those of furunculosis and other inflammatory lesions of the ear, a diagnosis will be made earlier. Dr. Spencer has brought out the type of lesion common in this region. The basal and the squamous cell type are those usually seen. At times there is parotid tumor, the mixed tumor of the parotid gland, and that secondarily involves the external ear. I have seen such tumors in the canal of the ear, causing considerable obstruction at that point.

As to the treatment, I feel that surgical diathermy is the procedure of choice. I have never observed a case in which the periosteum or the perichondrium was involved, and irradiation alone permanently cleared up the trouble. Most of the patients may stay well for a time and then the condition spreads along the periosteum or perichondrium, appearing a few years later farther along than the site of the primary lesion.

Epithelioma of the otic canal is a difficult disease to eradicate. On the other hand, as Dr. Mosher has brought out, the condition is often cleared up permanently if the diagnosis is made early, and in my experience surgical diathermy has been the best aid.

Dr. Thomas E. Carmody, Denver: I have a few slides that I should like to show, illustrating some cases. The first case is one similar to that to which Dr. New called attention, one of sarcoma that probably originated as a parotid tumor.

Dr. Frank R. Spencer, Boulder, Colo.: I have little to add.

My associates and I tried to treat these patients as thoroughly as we could. They were all treated in consultation with our full time radiologist. It is unfortunate that many of the tumors are not more radiosensitive than they are; if they were some of the fatalities could be avoided. Recently, after I had completed my paper, I saw an older man—a private patient and a professional man—with a small basal cell carcinoma near the helix. Early excision of that, followed by irradiation, will probably result in complete cure.

As I mentioned in the paper, all the patients were seen too late to get the brilliant results desired.

ACOUSTIC MOTOR REACTIONS

ESPECIALLY THE COCHLEOPALPEBRAL REFLEX

HANS STRAUSS, M.D.

CARNEY LANDIS, PH.D.

AND

WILLIAM A. HUNT, PH.D.

NEW YORK

In 1929 Strauss[1] published a monograph on startle (*Das Zusammenschrecken*). Subsequently, Landis, Hunt and others[2] published a series of articles dealing with the immediate response to the sound of a pistol shot. Parts of the reaction which we have called the "startle pattern" have been termed by various otologists the "cochleopalpebral reflex," the "auropalpebral reflex," the "acousticopalpebral reflex" or the "diffuse cochlear muscle reflex." Therefore, it seems worth while to call specific attention to certain of the otologic implications of our series of experiments.

The reaction pattern initiated by the sudden loud sound produced by firing a pistol has been studied by means of normal speed and ultra-high speed motion pictures. It has been clearly shown that the immediate reaction to the sound is always composed of movements forming a typical pattern: shutting of the eyes, characteristic distortion of the features, forward movement of the head, raising and drawing forward of the shoulders, abduction, forward elevation and inward rotation of the upper arms, bending of the elbows, pronation of the forearms, clasping of the hands, contraction of the abdomen, forward movement of the trunk and bending of the legs at the hips and knees. This pattern varies in the degree of its manifestation in various subjects and in

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From the New York State Psychiatric Institute and Hospital.

Financial support for this work has been given by the John and Mary Markle Foundation.

1. Strauss, H.: Das Zusammenschrecken: Experimentell-kinematographische Studie zur Physiologie und Pathophysiologie der Reaktivbewegungen, J. f. Psychol. u. Neurol. **39**:111, 1929.

2. Hunt, W. A., and Landis, C.: The Overt Behavior Pattern in Startle, J. Exper. Psychol. **19**:309, 1936. Hunt, W. A.: Studies of the Startle Pattern: II. Bodily Pattern, J. Psychol. **2**:207, 1936. Hunt, W. A.; Clarke, F. M., and Hunt, E. B.: Studies of the Startle Pattern: IV. Infants, ibid. **2**:339, 1936. Landis, C.; Hunt, W. A., and Page, J. D.: Studies of the Startle Pattern: VII. Abnormals, ibid. **4**:199, 1937.

various experiments on the same subject. Not always do all the separate elements of the pattern occur, but always some of them appear. This complicated reaction we have called the "startle pattern," and we believe that it is identical with the response designated by the otologic term "diffuse cochlear muscle reflex."

We have never found movements opposite to the movements described occurring as an immediate consequence of the shot. From this we conclude that descriptions reporting extension movements (Schaefer and Giesswein [3]) are based on results obtained with inadequate recording technic.

The cochleopalpebral reflex is a unit, indeed the most constant unit of the diffuse cochlear muscle reflex. This reflex has been the subject of many otologic discussions. Our experiments have definitely shown that this reflex invariably follows the sound stimulus in all persons having any remnant of hearing, except in certain epileptic persons, "clouded" psychotic persons and unconscious persons.

The otologic literature contains many reports stating that the reflex is not present in all persons with normal hearing (Cemach,[4] Falta,[5] Klestadt,[6] Schaefer and Giesswein [3]); that the subject can be completely adapted to it by repetition of the stimulus, or that it can be voluntarily inhibited (Belinoff,[7] Falta [5]). All these results are probably due to inadequate methods of observation and recording. Our experiments have shown that it is difficult to be certain of the occurrence of an eye blink by simple direct observation. Occasionally it is impossible to see the eye blink clearly in motion pictures taken either at normal or at four times normal speed. Only with high speed recording of from 200 to 300 exposures per second can the presence of the eye blink be certainly ascertained. We have shown that the eye blink sometimes starts within 0.005 second and is over in 0.015 second after the occurrence of the stimulus. Obviously, such a high speed reaction must frequently be missed when direct observation is relied on. Our results are in

3. Schaefer, K. L., and Giesswein, M.: Physiologie des Ohres, in Denker, O., and Kahler, O.: Handbuch der Hals-, Nasen und Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 6, p. 389.

4. Cemach, A. J.: Der akustische Blinzelreflex in der militärärztlichen Konstatierungspraxis, Wien. med. Wchnschr. **68**:2189, 1918.

5. Falta, M.: Zur Auslösung des cochlearen Lidreflexes, Monatschr. f. Ohrenh. **55**:319, 1921.

6. Klestadt, W.: Symptomatologie der Erkrankungen des N. VIII einschl. Leitung im Hirnstamm, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 4, p. 422.

7. Belinoff, G.: Das Mikrotympan, Wien. med. Wchnschr. **72**:2025, 1922.

accord with those of Poyet and Lallemant,[8] who, using mechanical registration, also found the cochleopalpebral reflex present in 100 per cent of their cases.

According to our observations the eye blink in response to a sound is always present in normal infants. (Our series did not include babies younger than 7 days of age, as did the studies of Alexander,[9] Berberich and Wiechers,[10] Catel,[11] Cemach,[4] Demetriades,[12] Kutvirt [13] and Voss.[14] Hence, to be perfectly certain of the presence of the eye blink reflex in an infant under 7 days of age and to eliminate the contradictions among the different experimenters, we should have to continue our experiments to include tests of a group of newborn infants.)

So, leaving the question open with respect to very young babies, we may say conclusively that the cochleopalpebral reflex is obtainable in all hearing persons except the few epileptic, psychotic or unconscious patients mentioned.

The reflex and the pattern are present even in persons who are practically deaf. We have examined a series of students from the New York School for the Deaf. All of them gave positive responses (and heard the pistol shot), though they were not able to hear speech and had only very little hearing as evidenced by their audiograms. The experiments on these subjects clearly showed that there was no relation between the degree of reaction and the amount of hearing remaining. This point has previously been debated by Wodak [15] in contradiction to Falta [5] and Foy.[16]. The degree of spread and intensity of reaction is probably determined by complicated circumstances, i. e., attention and tonus.

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8. Poyet, G., and Lallemant, M.: Recherches du reflexe cochleo-palpébraire par la méthode graphique, Arch. de méd. et pharm. mil. **68**:754, 1917.

9. Alexander, G.: Die Ohrenkrankheiten im Kindesalter, in von Pfaundler, M., and Schlossmann, A.: Handbuch der Kinderheilkunde, Leipzig, F. C. W. Vogel, 1931, vol. 7, p. 1.

10. Berberich, J., and Wiechers, A.: Zur Symptomatologie des Geburtstraumas Ztschr. f. Kinderh. **38**:59, 1924.

11. Catel, W.: Neurologische Untersuchungen und frühgeborenen Kindern, Monatschr. f. Kinderh. **38**:303, 1928.

12. Demetriades, T.: Der cochleopalpebrale Reflex bei Neugeborenen, Monatschr. f. Ohrenh. **55**:756, 1921.

13. Kutvirt, O.: Ueber das Gehör Neugeborener und Säuglinge, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **4**:166, 1911; **5**:249, 1912.

14. Voss, O.: Klinische und pathologisch-anatomische Folgeerscheinungen geburtstraumatischer Schädigungen des Felsenbeins, Monatschr. f. Kinderh. **34**: 568, 1926.

15. Wodak, E.: Ueber die Verwendbarkeit des durch die Baranysche Lärmtrommel erzeugten Lidreflexes zur Diagnose der Simulation, Monatschr. f. Ohrenh. **53**:23, 1919; Zur Auslösung des cochlearen Lidreflexes, ibid. **55**:591, 1921.

16. Foy, R.: Examen des surdités de guerre, Presse méd. **24**:595, 1916.

The occurrence of the eye blink in persons who are practically deaf proves (contrary to the opinion of Falta,[5] Gault [17] and Molinié [18] and in accordance with the opinion of Cemach,[4] Helsmoortel and Nyssen,[19] Mosso,[20] Wodak [15] and Wotzilka [21]) that it cannot be used to discover stimulation.

It has been held that in patients with very reduced hearing the eye blink may be the result not of the acoustic stimulation but of an extra-acoustic or purely sensory stimulation to the cornea or to the external ear (reflex of Fröschels [22] and Kisch [23]). However, we do not believe that our results can be explained in this manner. The distance between the pistol and the subject was too great to permit effective stimulation by air concussion. Finally, the exact differentiation between the activation of the eye blink by stimuli setting up impulses over the acoustic nerve and by stimuli affecting the trigeminal or other sensory pathways is much more difficult to envisage than earlier investigators have held. Every form of stimulation which is adequate for auditory sensation must cause movement within the middle or inner ear in order to set up impulses over the eighth nerve.

Transformations of energy sufficient to set up impulses leading to auditory sensation and perception must of necessity (in practically every case) be effective in setting up impulses over other sensory pathways, leading to sensation and perception of touch and movement.

The low energy level of most ordinary sensory stimulation leading to the usual forms of sound perception does not produce an eye blink. However, the limits of this general condition have still to be examined exactly. One method of doing this in man would be to examine patients with reduced hearing whose audiograms are available. With these persons auditory stimulation by sounds of various amplitudes and fre-

17. Gault, F.: Note sur l'utilisation du réflexe cochléo-orbiculaire pour la surdité, Presse méd. **24**:424, 1916.

18. Molinié, J.: Réflexes oculaires d'origine auditive, Rev. de laryng. **1**:385, 1916; cited by Mosso.[20]

19. Helsmoortel, J., Jr., and Nyssen, R.: Les réflexes cochléaires et leur valeur sémiologique, J. de neurol. et de psychiat. **30**:681, 1930.

20. Mosso, F. E.: I considetti "riflessi motori acustici," Ann. di med. nav. e colon. **31**:289, 1925.

21. Wotzilka, G.: Eine neue Art von Hörprüfung bei hochgradiger Schwerhörigkeit und Taubstummheit, Monatschr. f. Ohrenh. **57**:880, 1923.

22. Fröschels, E.: Eine Art von Hörprüfung bei hochgradiger Schwerhörigkeit und Taubstummheit, Monatschr. f. Ohrenh. **57**:1038, 1923; Ueber einen durch Schallreize ausgelösten Augenmuskelreflex bei Taubstummen, ibid. **61**:51 and 776. 1927.

23. Kisch, B.: Die Funktionsprüfung des Gehörorgans betreffende Beobachtungen, Deutsche med. Wchnschr. **1**:164, 1922.

quencies should be used. The occurrence or absence of the eye blink at each well defined stimulus should be recorded by an adequate objective method of registration. Such experiments would show whether the occurrence of the eye blink is concomitant with the distribution of the remnants of auditory sensibility, and it might be determined whether under such conditions the method can be used as an objective way of detecting deafness.

PRESENT STATUS OF THE PROBLEM OF OTOSCLEROSIS

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It is well known that certain characteristics and certain malformations are transmitted in particular families. The hereditary potentialities which lead toward them are believed to be carried in the germ cells in minute packets called genes, which unite in the fertilized ovum and persist in each cell division, modifying the cellular physiology of the animal and directing the growth along definite lines. In addition to the potentialities of the genes directed toward normal development, there are influences outside the cell which are necessary for and affect its growth and so are able to modify the ultimate individual outcome. These surrounding or environmental factors play an important role in the development of the organism, both in the intrauterine stage and after birth. To state how much of a person's resulting physical characteristics is due to heredity and how much to environmental factors is not at present possible; it is difficult enough when one deals with abnormalities but increasingly so when one deals with individual susceptibilities. The main facts, however, are clear. The basic structures which determine the direction of growth are passed on from one generation to the following. The individual status at any period is the resultant of the interaction of heredity and environment and is rarely the same in different persons. The hereditary factor can be modified by the environmental factors.

Recognition of this interplay and the use of adequate measures to modify an adverse hereditary tendency may produce an amelioration of its result. Thus the thyroid gland is essential to normal life, as it is the only organ capable of elaborating an iodine-containing hormone, necessary for the oxidation of proteins, fat and carbohydrates, as well as of influencing the excretion of calcium and magnesium. Thyroid insufficiency may lead to faulty nutrition and the formation of simple goiter. A hereditary factor best explains why in localities where goiter is prevalent one finds some families affected markedly by the disease, others comparatively little and others not at all; moreover, in one generation of a family of which all members have been exposed to the same surrounding influences one finds some with goiter and others free. The influence of environment is shown by a deficiency

of iodine in the food or in the drinking water in the locality where goiter is prevalent and by the observation that many persons with goiter are benefited by the administration of iodine.

One has to differentiate lesions, clinically identical, one of which is hereditary and the other produced by environmental factors. For instance, deaf-mutism may be hereditary (congenital deaf-mutism) or acquired as a result of disease after the fertilization of the ovum (acquired deaf-mutism). Congenital deaf-mutism is dependent on the developmental absence of central or peripheral nerve elements related to the internal ear. Though in one or more persons in a family or in one generation it may not appear, the power to produce the deficiency may be present awaiting, it is believed, a suitable combination of genes to call it forth. Marriages in families in which hereditary deaf-mutism occurs (especially consanguineous marriages) play an important part in its transmission; the children of the union of hereditary deaf-mutes may all be deaf-mutes. On the other hand, acquired deaf-mutism is not transmitted; it results from destruction of the auditory mechanism before the child has learned to talk, for instance, by intrauterine syphilis or by infectious diseases, such as meningitis and scarlet fever.

There are two generally recognized auditory deficiencies, early degeneration of the nerves and otosclerosis, in which an interplay of heredity and environment often can be traced.

NERVE DEAFNESS

In some families there is an inherited tendency of the auditory nerves to early degenerative changes, with resulting deficiency of hearing, usually about middle life. The loss of hearing is usually slow, and it may take years to bring about a marked deficiency. The auditory nerve can show a similar progressive change from injury by disease, such as syphilis, scarlet fever, influenza or arteriosclerosis; by drugs, such as quinine, or by persistent loud noises. In families in which there is the inherited tendency or susceptibility to early degeneration, environmental factors result in an earlier onset and often a more rapid degeneration.

OTOSCLEROSIS

Otosclerosis is a progressive deficiency of hearing which usually is noted early in adult life. Though in some cases no hereditary background can be shown, in others this element is so obvious that it cannot be excluded. That an environmental factor is of influence is demonstrated by the clinical history in many cases.

In any discussion on otosclerosis two questions must be considered:

1. What local changes seem hereditary, and what is the basic factor causing these changes?

2. Are there environmental factors influencing the occurrence of the condition as a clinical entity or its reappearance after what seems to be a period of quiescence?

Albrecht,[1] at the Third International Otorhinolaryngologic Congress, at Berlin, in 1936, stated clearly the view of the otologist concerning otosclerosis, ". . . there is a constitutional readiness on the part of the labyrinthine capsule to become transformed in structure. For the condition actually to occur an inciting environmental factor is necessary." With this statement geneticists will generally agree, but there is no agreement regarding the environmental factors which influence this transformation.

MORPHOLOGIC OBSERVATIONS

Otosclerosis is a change in that portion of the petrous bone which surrounds the inner ear (capsule of the labyrinth). It can be definitely recognized histologically as a sharply circumscribed area usually but not always bilaterally symmetric. By far the most common site is in front of the oval window, between the stapes and the cochlea; this area is so commonly affected that it is spoken of as the site of predilection. Though small at first, it may spread into the adjacent capsule and in this growth may involve the ligament of the stapes, diminishing its movement with resulting deficiency of hearing. More rarely it occurs at the innermost part of the internal auditory meatus and occasionally in the region of the semicircular canals. In it, islands of cartilage, so characteristic of the labyrinthine capsule, are absent. It appears as sharply defined porous bone. The picture suggests a slow absorption of the primary reticular capsular bone, followed by a slow deposition of altered bone; if the former predominates the pores are large, if the latter predominates the pores are small, with occasional hyperostosis or exostosis. The pores vary greatly in size and shape and communicate more or less freely. They contain blood vessels and fatty marrow. At the periphery, where the nodule grows, the spaces are usually small. Osteoblasts are numerous, but only an occasional osteoclast is noted. Vessels pass from the nodule into the normal bone of the capsule but not in the reverse direction.[2] The reticular bone of the normal capsule ends abruptly at the nodule and sharply marks it off. Its size varies greatly; it may extend, for instance, from the endosteum of the vestibule to the mucoperiosteum of the middle ear and around the cochlea; it may be a small isolated nodule in the capsule, not involving the ligament of the stapes.

1. Albrecht, W.: Die Bedeutung der Konstitution für Hals, Nase und Ohr. Allgemeiner Überblick, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:295, 1936.

2. Wilson, J. G., and Anson, B. J.: Form and Structure of an Area of Otitic Sclerosis in the Temporal Bone of an Adult, Arch. Otolaryng. **18**:291 (Sept.) 1933.

As fixation of the stapes is the chief cause of the deficiency of hearing, it is this phase which has chiefly attracted the otologist. The hereditary factor was early recognized. Observations in clinical cases compelled many physicians to believe that environmental factors play no insignificant part in the onset and development of the condition. Regarding these environmental factors there is no uniformity of opinion. The most marked uniformity is on the point that in predisposed persons pregnancy may at times precipitate its occurrence or increase a deficiency of hearing already present. There are some authors who have expressed the belief that catarrh of the middle ear in young adults of otosclerotic families results in an early onset of the symptoms.

To determine the underlying cause of the morphologic change numerous hypotheses have been advanced, from which the following may be selected:

1. Wittmaack [3] concluded from morphologic examination of clinical material that the otosclerotic process is due to a damming back of the flow of venous blood in the labyrinthine capsule, with consequent stagnation in the smaller vessels and osteoporosis. He stated that he had produced a similar condition in the labyrinthine capsule of chickens by experimentally produced stagnation. His attempts to produce corresponding biologic conditions in monkeys were unsuccessful, and he concluded from his anatomic study that the mammalian venous circulation is such that an analogous damming back cannot be accomplished experimentally. He expressed the opinion that in persons with a hereditary predisposition otosclerosis may develop without manifestation of marked stasis in the morphologic picture. Not all histologists agree that his experimentally produced change is identical with otosclerosis. But even should it be agreed that a damming back may produce stasis with resulting porous bone, the causative factor remains unexplained.

2. Mayer [4] stated that otosclerosis is a form of reaction to mechanical influences or tensions, that is, a mechanical traumatism. It is a hyperplasia typically located in a region where a synchondrosis or joint, the fissula ante fenestram, exists. This synchondrosis indicates an area where there is a need of compensation for tensions between the cochlear part in front and the vestibular behind. It is a constant feature in the embryo and in the newborn infant but tends to disappear with age. In the periosteal aspect of this joint the otosclerosis commences as a small area of atypical bone such as is known to occur in similar positions in other parts of the body. From these areas of

3. Wittmaack, K.: Die Ursache der Otosklerose: Ein Vorschlag zur ursächlichen Behandlung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:150 (June 26) 1931.

4. Mayer, O.: Pathology of Otosclerosis, J. Laryng. & Otol. **43**:843 (Dec.) 1928.

atypical bone otosclerotic foci develop. Since the otosclerotic foci occur most frequently only in the periosteal portion of the synchondrosis by the ossification and obliteration of this portion the compensation of the tension through the synchondrosis is inhibited and the bone then laid down, instead of being solid periosteal bone, is otosclerotic bone, which is better able to meet the mechanical strain.

Against the hypothesis of synchondrosis it can be shown that the formation of the fissula is not comparable to that of a synchondrosis. Bast [5] has pointed out that the fissula forms from a dedifferentiation of precartilage in the area between the stapes and the cochlea into loose mesenchymal tissue; this, extending from the vestibule to the middle ear, later becomes more fibrous, and the surrounding precartilage changes into cartilage and later into bone. A synchondrosis forms as a progressive change of embryonic connective tissue into precartilage and then to cartilage between two areas of bone. Again, if relief of tensions in this area is so important, it is remarkable that similar fissulae are not found in other mammals; not even a trace can be seen in the fetuses of many mammals which have been examined.[6] Moreover, it is not generally agreed that the otosclerotic area appears first in the periosteal bone.

3. Gray [7] expressed the opinion that the essential causative factor in otosclerosis is a degenerative process affecting the whole organ of hearing, from the external auditory meatus to the cerebral cortex. While several environmental conditions may play a part in its causation, he stated that the basic cause is a gradually increasing defect in the reflex vasomotor mechanism which governs the nutrition of the organ of hearing as a whole. Since the stimulus which excites the vasomotor mechanism of audition is sound and sound alone, the vestibular apparatus is unaffected. The preponderance of women among patients with otosclerosis is the result of the greater instability of their vasomotor system and the more frequent disturbances to which it is exposed. He stated that the deafness of otosclerosis bears little relation to the extent of the disease in the bone. The deafness may be severe when the stapes is hardly fixed at all. The severity of the tinnitus bears no relation to the duration of the disease. The deafness is to a larger extent functional and is the result of the insufficient supply of blood to all the nerve structures concerned in the perception of

5. Bast, T. H.: Osteogenesis of the Human Periotic Capsule, Arch. Otolaryng. **10**:459 (Nov.) 1929.

6. Wilson, J. G.: Fissula Ante Fenestram and the Adjacent Tissue in the Human Otic Capsule, Acta oto-laryng. **22**:382, 1935.

7. Gray, A. A.: Otosclerosis Problem: Including Reports of Two Cases Pathologically Examined (Dalby Memorial Lecture), Proc. Roy. Soc. Med. **27**: 1179 (July) 1934.

sound. The pathologic results seen are largely observed in the part which is subject to constant strain, namely, anterior to the stapes.

4. Fraser [8] stressed the environmental factor, especially the influence of catarrhal otitis media and other infectious conditions in the middle ear, in the genesis of otosclerosis in persons with a hereditary tendency. In support of this view he gave examples in which otosclerosis had manifested itself after inflammation of the middle ear. Against this it has been argued that in many cases there are no signs of inflammation, present or past. Gray, though not accepting Fraser's hypothesis, acknowledged that in some cases conditions in the nasopharynx or in the middle ear may be the immediate factor in the onset of otosclerosis in predisposed persons.

5. Several authors have expressed the opinion that the environmental factor is to be sought in a nutritional change. They pointed out that the endochondral layer of the capsule has been built up from a weak mesenchyme, a belief suggested by the association of otosclerosis with fragilitas ossium and blue scleras. This has resulted in a medulla rich in fiber, which gradually is transformed into bone, partly lamellar, showing a mosaic structure. Nager and Meyer [9] suggest linking the condition with Paget's disease and other diseases of bone under the general term osteodystrophia.

Objections can be urged against all these hypotheses, but through all there runs the basic idea of a congenital tendency or deficiency. Therefore, fundamental to an understanding of this disease is a knowledge of the development of the periotic capsule and how it differs from normal bone elsewhere in the body.

The bony periotic capsule develops, as does bone everywhere, from primitive connective tissue, first into cartilage and later into bone. The cartilage becomes covered externally by a dense connective tissue, the perichondrium, from which is formed the periosteal bone, similar to that found in the long bones of the skeleton. The cartilage surrounding the membranous labyrinth is displaced by primary endochondral bone in which is embedded intrachondrial bone or cartilage islands, a structure intermediate between bone and cartilage. This primary bone is characterized by its weblike strands, with the bone cells laid irregularly but in a direction more or less longitudinal. Elsewhere in the skeleton, e. g., in long bones, this primary bone is replaced by lamellar bone around haversian canals. This primary bone in the labyrinthine

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8. Fraser, J. S., and Muir, R.: Pathology of Otosclerosis, J. Laryng., Rhin. & Otol. **31**:465 (Nov.) 1916.

9. Nager, F. R., and Meyer, M.: Die Erkrankungen des Knochensystems und ihre Erscheinungen an der Innenohrkapsel des Menschen, Berlin, S. Karger, 1932, p. 230.

capsule persists, and haversian lamellas are few. In short, the fully formed capsule consists of a persisting primary bone, reticular and not lamellar. Compared with bone elsewhere, it is immature bone.

In the fetus, in the area between the stapes and the cochlea, there is a minute communication between the vestibule and the middle ear, filled with loose connective tissue and surrounded by cartilage, called the fissula ante fenestram. This fissula usually disappears, and the anatomic structure in this area passes to a formation similar to that elsewhere in the periotic capsule. But the fissula may not disappear, and the normal progress may be interrupted and show developmental irregularities spreading into the surrounding area. For instance, the loose connective tissue may persist from fetus to adult (70 years); the cartilage normally present in the fetus may increase and persist during life, irregular in size and location. There are often marrow spaces which show invasion by cartilage. Abnormal bone may be present irregular in outline, varying in amount, sharply separated from the tissue in which it lies and showing a lamellar structure around blood vessels or marrow spaces. In short, not only is the bone of which the periotic capsule is composed of unique character, but, when compared with bone elsewhere in the body, it remains during life largely in a primitive stage yet capable of achieving greater maturity. It is hard to determine the significance of the unique character of this capsular bone and of this diversity of tissue within the fissular area. If it is considered that the final goal to which the periotic capsule is directed is bone of a type which is partly primary, partly lamellar and partly intrachondrial, it is obvious that frequently in this area the tissues do not reach their fulfillment. One finds fibrous tissue, cartilage, enlarged marrow spaces and even small sclerotic masses surrounding enlarged haversian canals, apparently indiscriminately disposed between the stapes and cochlea.

Age is not the only factor, for this area shows individual differences in configuration and histologic content; for instance, the fibrous tissue may persist from fetus to adult (57 years), and cartilage, normally present in the young and later disappearing, may persist. The usual development of the primitive tissue, in this area, into cartilage, primary bone and intrachondrial bone, may be interrupted at any stage, and the relative increase of any one may be greater than normal. What factor or factors determine this interruption of normal growth I am not prepared to say, but I regard this area as in a state of disequilibrium, and the appearance of dilated vessels inclines me to believe that the vascular factor plays an important part in the subsequent course. The area, especially that part related to the fissula ante fenestram, is an area of anatomic variability, in which individual peculiarities as well as age are

factors.[10] It is in this area that otosclerosis usually occurs; even when no otosclerosis is present, small areas of sclerotic bone, surrounding dilated canals with blood vessels and marrow, are not uncommon. The anatomic variability offers an assured basis on which to proceed to a hypothesis:

Given anatomic variabilities, it may be and probably is the fact that otosclerosis can result from any of several secondary factors, especially those affecting the blood supply. The hypothesis is in keeping with clinical observations. The morphologic appearance points to alteration in capillary circulation, and since the findings of Anson and me [2] lead to the belief that the main source of arterial supply and probably of venous drainage in this area is associated with vascular circulation in the middle ear one readily thinks of trouble in the middle ear as a possible source of this condition.

I believe it is not justifiable to write down otosclerosis as a degenerative process; rather, it ought to be considered an inherited weakness with various degrees of susceptibility to environmental influences, internal or external, which may be interactive. This suggests a line of inquiry which has been much neglected and has been at no time the object of a systematic study. For it is with such influences that the clinician may hope to deal more or less effectively and ascertain under what conditions they act and with what consequences.

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10. Wilson.[6] Anson, B. J., and Martin, J., Jr.: Fissula Ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303 (March) 1935.

AUDITORY THRESHOLD OF UNPLEASANTNESS IN NORMAL AND IN HARD OF HEARING SUBJECTS

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In tests of auditory acuity involving persons with deficient hearing I have found—as have others previously—that the threshold is critical. Persons who are hard of hearing may not perceive a tone at one stage of amplification, but when it is intensified the minimum degree possible with an ordinary audiometer, it appears quite loud to them. Examination of normal persons does not show this phenomenon, but rather, as Fowler put it, "the threshold is a little indefinite without repeated search." [1]

Reger has also reported this finding, commenting that the threshold of those whose hearing is subnormal is more exact and more critical, and further that

> . . . the more pronounced the hearing loss the louder the auditory sensation when the intensity is increased a given number of db above the threshold. Second, the magnitude of the auditory sensation seems to increase more rapidly at intensity levels immediately above the threshold. This means that an increase in intensity of 5 db of a sound which is 2 db above threshold would sound relatively louder than a 5 db increase in intensity of a sound which is, e. g., 20 db above threshold.[2]

Fowler said also: "Comparative loudness showed that loudness of tones (near 4096) of from 20 to 25 sensation units above the threshold in the left (non-affected) ear appeared the same loudness as 5 sensation units above the threshold in the right (affected) ear.[1]

Fletcher has pointed out that, since all sounds of speech must lie in the band between the threshold of hearing and the threshold of pain and since the band between the two is much reduced for persons with hypacusis, there exists but a narrow ribbon through which the sub-

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Part of a thesis submitted to the faculty of the Graduate School of the University of Wisconsin in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Speech Pathology.

1. Fowler, E. P.: Marked Deafened Areas in Normal Ears, Arch. Otolaryng. **8**:151 (Aug.) 1928.

2. Reger, S. N.: Differences in Loudness Response of the Normal and Hard-of-Hearing Ear at Intensity Levels Slightly Above the Threshold, Ann. Otol., Rhin. & Laryng. **45**:1029 (Dec.) 1936.

normal subject may receive useful auditory stimulation.[3] However, even to the normal subject, sounds of sufficient intensity may become unpleasant before actually becoming painful. If it is true that the loudness of sounds increases more rapidly when the intensity is increased for persons with hypacusis than for normal persons, then may not this band between the threshold of hearing and unpleasantly loud sounds be smaller for the former than the one suggested by Fletcher? Would not any lowering of the upper limits of that range of hearing which is pleasant to the subject tend to limit the usable range? To answer these questions I have determined the amount of amplification above the threshold of hearing which is necessary before the sound becomes unpleasantly loud for normal subjects—using pure tones—and have made the same determination on hypacusic subjects.

The persons used in this investigation were from two groups: the first, 25 unselected students at the University of Wisconsin with normal hearing, 10 men and 15 women, ranging in age from 23 to 35; the second, 25 students at the State School for the Deaf, Delavan, Wis., 13 boys and 12 girls, ranging in age from 11 to 21, with an average loss of hearing of 46.3 decibels and a percentage loss of 45.98.[4] In testing members of the first group, whose hearing was normal, a Western Electric 2-A audiometer was used, while in testing members of the second group, whose hearing was subnormal, a Western Electric 6-A audiometer was used because of the increased range of intensity available. The procedure was as follows: In a relatively quiet room an audiogram was made for each subject, covering the range from 64 cycles to 8192 cycles, the opposite ear being unmasked, since my investigation in masking,[5] as well as those of others, indicates that when both ears are relatively similar in acuity masking has little or no effect.[6] After the completion of the test of acuity, the intensity of each tone was increased until the subject considered it unpleasantly loud; this procedure was followed three times if the results were consistent, but further trials were made if they proved inconsistent. When the subject found the point at which further increases in intensity produced an unpleasantly loud sound, a point 5 decibels above and one 5 decibels below this point were chosen, and he was then asked to indicate which of these three levels seemed most pleasant to him. Through these tests the borderline between that which was relatively pleasant and that which tended to become unpleasant so far as pure tones are concerned was determined for both groups.

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3. Fletcher, H., in discussion on Macfarlan, D.: The Report of the Committee Appointed to Survey the Instrument Aids to Hearing, Volta Rev. **29**:629, 1927.

4. This percentage was established by the method given by West, according to which the three highest frequencies, 2048, 4096 and 8192 cycles, are doubled and the sum divided by 11. (West, R.; Kennedy, L., and Carr, A.: The Rehabilitation of Speech: A Textbook of Diagnostic and Corrective Procedures, New York, Harper & Brothers, 1937 p. 397.)

5. Ansberry, M.: Auditory Deficiencies of Acuity and Their Correction with Prosthetic Appliances, Doctoral Dissert., University of Wisconsin, June 1937, p. 32.

6. Bunch, C. C.: Auditory Tests in Cases of Marked or Complete Unilateral Deafness, Ann. Otol., Rhin. & Laryng. **40**:748 (Sept.) 1931. Meissner, H.: Methoden der Hörprüfung eines Ohres, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:415, 1936.

TABLE 1.—*Differences Between the Thresholds of Hearing and of Unpleasantness in Normal and in Hard of Hearing Subjects*

By Subject

Normal Subjects				Hard of Hearing Subjects			
	Average in Decibels for Eight Frequencies from 64 Cycles to 8,192 Cycles				Average in Decibels for Eight Frequencies from 64 Cycles to 8,192 Cycles		
Subject	Right Ear	Left Ear	Average	Subject	Right Ear	Left Ear	Average
1	23.12	26.25	24.68	1	15.63	21.25	18.44
2	33.75	31.87	32.81	2	4.38	13.125	8.75
3	15.63	11.25	13.44	3	11.875	11.27	11.57
4	19.37	18.75	19.06	4	6.87	15.00	10.93
5	25.00	31.25	28.12	5	9.37	11.25	10.31
6	16.25	15.00	15.62	6	18.75	14.35	16.56
7	16.87	11.75	14.31	7	18.12	19.37	18.74
8	33.12	37.50	35.31	8	7.50	13.75	10.62
9	10.62	7.50	9.06	9	7.50	5.62	6.56
10	25.00	20.00	22.50	10	8.13	5.00	6.56
11	19.37	18.12	18.74	11	10.00	9.37	9.68
12	20.62	11.87	16.24	12	9.37	11.25	10.31
13	47.50	44.41	45.95	13	7.50	6.25	6.875
14	26.87	24.37	25.62	14	3.12	15.63	9.375
15	49.37	46.25	47.81	15	7.50	10.00	8.75
16	27.50	20.62	24.06	16	14.37	4.38	9.375
17	20.62	21.87	21.24	17	5.625	4.375	5.00
18	11.87	16.25	14.06	18	25.00	20.00	22.50
19	25.63	22.50	24.06	19	10.62	6.25	8.435
20	27.50	29.37	28.43	20	16.25	18.12	17.18
21	31.25	46.87	39.06	21	8.12	5.00	6.56
22	21.87	15.62	18.74	22	9.38	10.00	9.69
23	30.00	31.25	30.62	23	13.75	9.37	11.56
24	20.00	15.00	17.50	24	11.87	10.00	10.93
25	33.75	37.50	35.62	25	5.00	8.75	6.87
Average	25.30	24.50	24.90	Average	10.60	11.15	10.887

By Frequency

Frequency	Right Ear	Left Ear	Average	Frequency	Right Ear	Left Ear	Average
64	19.2	22.0	20.6	64	11.2	11.0	11.1
128	23.2	23.2	23.2	128	11.0	10.6	10.8
256	26.6	26.4	26.5	256	10.6	12.4	11.5
512	25.6	24.8	25.2	512	9.8	11.0	10.4
1024	30.4	29.0	29.7	1024	12.4	11.4	11.9
2048	29.0	26.8	27.9	2048	10.4	11.8	11.1
4096	27.0	25.4	26.2	4096	10.4	11.6	11.0
8192	21.6	19.4	20.5	8192	9.6	9.8	9.7

TABLE 2.—*Difference Between the Threshold of Unpleasantness in Normal and in Subnormal Subjects*

Frequency	Average Difference Between the Thresholds of Hearing and of Unpleasantness		Standard Deviation of the Averages		Probable Error of Averages		Difference Between the Two Averages	Probable Error of Difference	Difference Divided by Probable Error of Difference	Chances in 100
	Normal	Subnormal	Normal	Subnormal	Normal	Subnormal				
64	20.6	11.1	8.0	8.1	0.76	0.75	9.5	1.07	8.9	100
128	23.2	10.8	9.4	7.8	0.90	0.74	12.4	1.18	10.5	100
256	26.5	11.4	10.3	7.0	0.98	0.67	15.1	1.19	12.7	100
512	25.2	10.4	11.0	7.3	1.05	0.70	14.8	1.26	11.8	100
1024	29.7	11.9	12.5	8.6	1.19	0.82	17.8	1.44	12.3	100
2048	27.9	11.1	13.3	7.5	1.27	0.71	16.8	1.46	11.5	100
4096	26.2	11.0	14.7	8.8	1.40	0.84	15.2	1.64	9.3	100
8192	20.5	9.7	14.2	9.7	1.35	0.92	10.8	1.64	6.6	100

A summary of results is given in table 1 and shown graphically in charts 1 and 2. In table 2 is found the statistical relation between the two tests. In every case there is a statistically reliable difference between the two averages.

The threshold of unpleasantness is not the threshold of pain—it is merely that point at which sounds, although tolerable without actual pain, are no longer pleasant. In this connection, one may observe that this threshold of unpleasantness bears a direct relationship to the

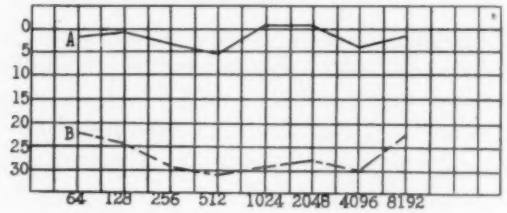

Chart 1.—Thresholds of hearing and of unpleasantness in subjects with normal hearing. *A*, threshold of hearing; *B*, threshold of unpleasantness.

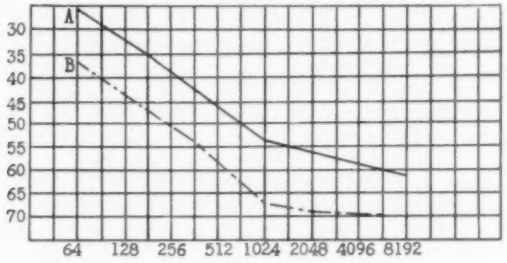

Chart 2.—Thresholds of hearing and of unpleasantness in hard of hearing subjects. *A*, threshold of hearing; *B*, threshold of unpleasantness.

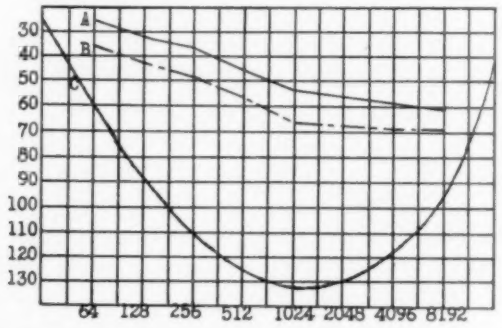

Chart 3.—Thresholds of hearing, unpleasantness and pain. *A*, threshold of hearing; *B*, threshold of unpleasantness; *C*, threshold of pain.

threshold of acuity, as shown by charts 1 and 2, but has no such contour as the threshold of pain, as is shown in chart 3.[7]

7. Adapted from the Western Electric Audiogram.

The results of this investigation indicate that for persons with normal hearing a tone of 64 cycles may be increased 20.6 decibels above the threshold before it becomes unpleasantly loud, while for the person with hypacusis an increase of but 11.1 decibels has the same effect. For 128 cycles the increase may be 23.2 decibels, as compared with 10.8; for 256 cycles, 26.5, as compared with 11.4; for 512 cycles, 25.2 and 10.4; for 1024 cycles, 29.7 and 11.9; for 2048 cycles, 27.9 and 11.1; for 4096 cycles, 26.2 and 11.0, and for 8192 cycles, 20.5 and 9.7. Over the entire range that extends from 64 cycles to 8192 cycles, the average difference between the threshold of acuity and the threshold of unpleasantness for the normal person is 24.9 decibels, whereas that for the person with hypacusis is 10.887 decibels, which permits less than half as great an increase.[8]

The range of usable, enjoyable sounds for the person with subnormal acuity is therefore greatly reduced—reduced much more than is apparent by a mere determination of the threshold of hearing. This fact may well indicate one of the greatest difficulties to be overcome in the fitting of hearing aids, since an amplifier which responds more readily at one frequency than at another will break through this narrow band of pleasant sounds at that point and cause the user to decrease the overall volume. This change in volume, in turn, causes the entire loss of some of the weaker sounds of speech, since they may lie in less responsive bands.

8. In 11.5 per cent of the hard of hearing subjects, any increase above the threshold was considered unpleasant.

THROMBOSIS OF THE LATERAL SINUS

A SURVEY OF CURRENT OPINION AND RECORDS

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While thrombosis of the lateral sinus is the intracranial complication most frequently encountered after operations for mastoiditis, the number of cases observed by the average otologist is so small that he feels obliged to draw on the larger collective experience of his confrères in formulating criteria for the management of this serious condition. When he consults the literature, he finds many excellent reports of cases and the expression of a wide diversity of opinion regarding the efficacy of the surgical procedures used to combat thrombosis of the lateral sinus. It is difficult, if not impossible, to draw definite conclusions, for there are records which seem to substantiate each contrasting opinion.

Since the individual series of cases reported are, as a rule, small, it occurred to me that to obtain the recent records from various parts of the country on the incidence of thrombosis of the lateral sinus following operations for mastoiditis and the outcome might yield valuable information concerning this complication. So far as I have been able to determine, such records have not been assembled. A letter to the medical department of the Metropolitan Life Insurance Company brought the reply that that organization has no figures on the incidence of thrombosis of the lateral sinus following mastoid operations in various parts of the country and knew of no one who could furnish such information.

Approximately a thousand questionnaires were sent to leading otologists and hospitals in the United States and Canada. To this inquiry three hundred and forty-three replies were received. These came from physicians and hospitals in thirty-nine states, Hawaii and the District of Columbia, as well as five provinces in Canada (chart).

Of this number, one hundred and seventy-eight gave actual figures concerning the number of operations on the mastoid and the number of cases in which thrombosis of the lateral sinus occurred. Nineteen more gave information concerning the number of cases of thrombosis of the lateral sinus or the percentage incidence of the complication without

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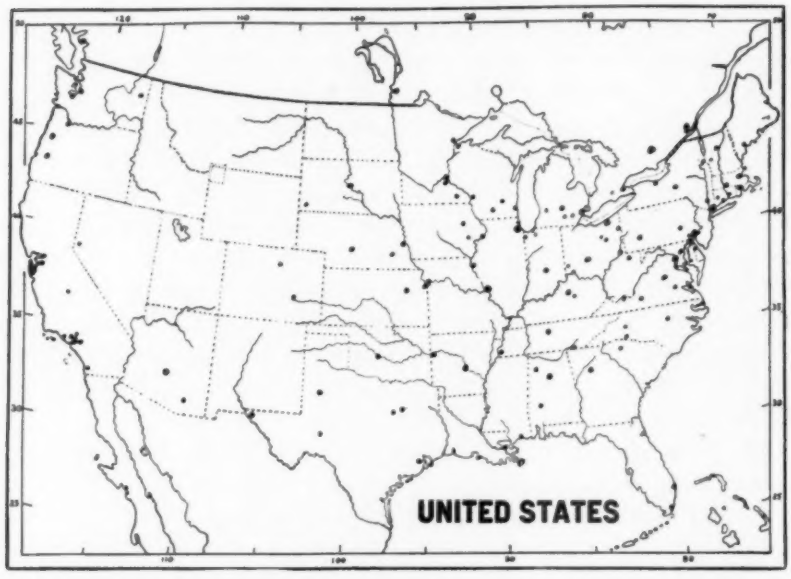
the actual number of cases either of operation or of thrombosis. Only ninety-two included information concerning the number of deaths from thrombosis of the lateral sinus. Ninety-one letters contained some comment on diagnosis, methods of treatment or clinical details. Forty-three of the replies contained information regarding the number of patients operated on and the type of operation used.

In this study an attempt is made to analyze and catalogue the various facts and factors concerning thrombosis of the lateral sinus contained in this correspondence. The discussion is augmented by citations from the American literature of the last ten years. No attempt



Map showing places from which information on the incidence of thrombosis of the lateral sinus was obtained.

has been made to collect an exhaustive history of the subject or to include a complete bibliography. A large proportion of the articles in the current literature consist of collections of cases, and it is beyond the scope of this study to refer to them all directly. The purpose is rather to glean from this material such figures, facts and opinions regarding thrombosis of the lateral sinus as seem representative of present otolaryngologic practice in America.

INCIDENCE

The one hundred and seventy-eight replies that furnished exact figures as to the number of operations on the mastoid and the number

of cases of thrombosis of the lateral sinus yielded a total of 59,850 cases in which the mastoid was operated on, in 1,556 of which thrombosis of the lateral sinus occurred, the incidence being 2.6 per cent. The incidence in the individual replies varied between 0 and 33 per cent. The complete data are shown in table 1.

In comparing these records a number of factors must be kept in mind. In material collected in this manner it is impossible, of course, to get figures from a large number of sources that can be justly compared. When complete details are not available, the figures themselves often do not give a reliable picture. In the tabulation of these data it is obvious that the different criteria accepted in different places for the diagnosis of thrombosis of the lateral sinus will affect the incidence. Some contributors excluded cases in which the patient was in a hopeless condition when admitted; in other series these were obviously included, since the information furnished merely stated the total number of mastoid operations and the total number of cases of thrombosis of the lateral sinus. In some institutions the records are such that thrombosis of otitic origin can easily be separated from other conditions, and in others all cases of thrombosis are lumped together.

Other factors which affect the incidence of thrombosis of the lateral sinus include the type and virulence of the infecting organism, the age of the patient and his economic status, i. e., whether he is a charity or a private patient. After certain virulent infectious epidemics the incidence of thrombosis of the lateral sinus may be higher than for aural infections generally. The disease is more common in children, and hence a children's hospital might show a higher incidence than a general hospital. It seems to be true that the incidence is higher in charity hospitals than in those caring only for private patients, because the patients who come to the former have often been neglected until the aural infection was extremely far advanced. On the other hand, well known specialists who practice almost entirely among referred patients —and these often in the most desperate condition—reported the highest incidences of thrombosis of the lateral sinus. It may be, too, that their larger experience with this condition has caused them to be more alert in the diagnosis.

It also seems to be true that the more recent figures indicate a higher incidence of thrombosis of the lateral sinus than was reported formerly. This is brought out nicely in the report of Meltzer on the cases observed at the Massachusetts Eye and Ear Infirmary, Boston. From 1921 to 1926 the incidence was 2.5 per cent in 2,496 cases of mastoiditis and from 1927 to 1932 the incidence was 3.9 per cent in 2,465 cases. Whether better diagnosis was the only factor here is difficult to say.

TABLE 1.—*Incidence of Thrombosis of the Lateral Sinus in Mastoiditis*

Location	Source	Period Covered by Report, Years	Cases of Mastoiditis	Cases of Sinus Thrombosis	Incidence of Sinus Thrombosis, %
Alabama					
Birmingham	Hillman Hosp.	2	33	0	0
	Pope	7	Ca. 125	3	Ca. 2.4
Jasper	Wickliffe	14	109	0	0
Selma	Kirkpatrick	?	Ca. 200	4	2
Arizona					
Phoenix	Thoeny	10	131	5	3.8
	Schwartz and Melton	10	?	3	?
Tucson	Comer	?	20 per yr.	0	0
	Patterson	1	17	0	0
Arkansas					
Ft. Smith	Moulton	?	?	1	?
Little Rock	Mahoney	5	184	8	4.3
California					
Fresno	Fresno County Gen. Hosp.	3	32	10	31.25
Glendale	Donohoe	1	?	9	?
Long Beach	Horner (3 hosps.)	5	330	16	4.8
Los Angeles	Children's Hosp.	10	582	32	5.4
Oakland	Beaudoux	30	Ca. 500	0	0
Pasadena	Dysart	5	19	0	0
	Roberts	25	70	4	5.7
	Wimp	13	Ca. 75	0	0
San Diego	San Diego Co. Gen. Hosp.	10 mo.	18	?	?
	U. S. Naval Hosp.	2	6	?	?
San Francisco	Children's Hosp.	13	1,192	27	2.3
	San Francisco Hosp.	11	375	4	1.1
	U. S. Marine Hosp.	7	16	0	0
	St. Luke's Hosp.	8	175	1	0.6
	Morrison	9	180	8	4.2
Santa Barbara	Cottage Hosp.	5	41	1	2.4
Whittier	Smith	?	10	0	0
Colorado					
Denver	Children's Hosp.	1	64	2	3.1
	Colorado Gen. Hosp.	5	69	1	1.4
	Beyer	?	Ca. 160	1	Ca. 0.6
Pueblo	Corwin Hosp.	4	28	0	0
Connecticut					
Bridgeport	Bridgeport Hosp.	9	164	3	1.8
New London	Robison	5	107	1	0.9
New Haven	New Haven Hosp.	3	117	1	0.9
Delaware					
Wilmington	Mayerberg	?	?	5	?
District of Columbia					
Washington	Episcopal Eye, Ear and Throat Hosp.	5	731	27	3.7
	Walter Reed Gen. Hosp.	5	142	0	0
	Washington San. and Hosp.	7	14	1	7.1
Florida					
Jacksonville	Knauer	10	Ca. 140	5	Ca. 3.6
Miami Beach	Repass	5	?	1	?
West Palm Beach	Sayad	5	65	2	3.1
Georgia					
Atlanta	Grady Hosp.	3	Ca. 35	2	?
	Warren	8	81	3	3.7
Illinois					
Chicago	Children's Mem. Hosp.	14½	550	13	2.4
	Grant Hosp.	4	99	2	2
	St. Luke's Hosp.	7	260	6	2.3
	Pollack	9	Ca. 900	?	Ca. 2
Evanston	Evanston Hosp.	2	44	3	6.8
Quincy	Stevenson	?	200	4	2
Indiana					
Hammond	Kuhn	?	Ca. 900	14	Ca. 1.6
Indianapolis	University Hosps.	3	284	8	2.7
	Methodist Hosp.	5½	105	6	5.7
	Indianapolis City Hosp.	2	154	4	2.6
South Bend	Epworth Hosp.	5	90	2	2.2
	St. Joseph Hosp.	5	49	0	0
Mishawaka	St. Joseph Hosp.	5	59	14	23.7

TABLE 1.—*Incidence of Thrombosis of the Lateral Sinus in Mastoiditis—Continued*

Location	Source	Period Covered by Report, Years	Cases of Mastoiditis	Cases of Sinus Thrombosis	Incidence of Sinus Thrombosis, %
Iowa					
Cedar Falls	Grant	7	32	0	0
Davenport	Hofmann	?	150	?	Ca. 5
Iowa City	University Hosp.	1	Ca. 250	8	Ca. 3.2
	Albright	?	111	1	0.9
Kansas					
Kansas City	Bell Mem. Hosp.	2	55	3	5.5
Topeka	Reed	10	?	?	Ca. 4
Kentucky					
Lexington	? Hosp.	5	128	3	2.3
	? Hosp.	4	102	6	5.9
Louisville	City Hosp.	5	96	6	6.2
Louisiana					
New Orleans	Hotel Dieu	5½	156	4	2.6
	Fuchs	1½	130	1	0.8
Maine					
Portland	Maine Gen. Hosp.	4½	240	4	1.7
Maryland					
Baltimore	Johns Hopkins Hosp.	20	Ca. 1,000	30	Ca. 3
	Union Mem. Hosp.	19	163	1	0.6
	Cohen	?	138	2	1.4
Massachusetts					
Boston	Boston City Hosp.	5	489	17	3.5
	Mass. Eye and Ear Infirmary	15	6,348	202	3.2
	Tobey (private)	2	127	10	7.9
Worcester	City Hosp.	10	323	7	2.2
	Belmont Hosp.	3	?	5	?
Michigan					
Ann Arbor	University Hosp.	8	638	10	1.6
Detroit	Children's Hosp.	5	1,408	14	1
	Ford Hosp.	10	601	10	1.7
	Herman Kiefer Hosp.	5	235	15	6.4
Grand Rapids	Blodgett Mem. Hosp.	5	145	2	1.4
	Butterworth Hosp.	4	182	4	2.2
Lansing	Ellis	7	Ca. 100	4	Ca. 4
Jackson	Hardie	5	18	0	0
Minnesota					
Minneapolis	Minn. Gen. Hosp.	6	284	0	0
	Shriner's Hosp.	10	5	0	0
	Eye, Ear, Nose and Throat Clinic	9	129	0	0
	Spratt	28	251	21	8.4
Duluth	St. Luke's Hosp.	5¾	145	2	1.4
	St. Mary's Hosp.	2	36	2	5.6
Rochester	Mayo Clinic	?	933	88	9.4
St. Paul	Ancker Hosp.	8	?	10	?
Mississippi					
Gulfport	McWilliams	6	24	1	4.2
Missouri					
Kansas City	Research Hosp.	4½	?	3	?
St. Louis	Barnes and St. Louis Children's Hosp.	20	1,420	52	3.7
	Jewish Hosp.	2	82	3	3.7
	Westlake	30	?	70	?
Nebraska					
Grand Island	Grand Island Clinic	?	25	2	8
Lincoln	Brooks	12	98	3	3.1
	Hompes and Peterson	10	277	10	3.6
Nevada					
Reno	Fuller	16	Ca. 150	0	0
New York					
New York	Bellevue Hosp.	3	825	12	1.5
	Manhattan Eye, Ear and Throat Hosp.	12	7,012	170	2.4
	N. Y. Eye and Ear Infir.	11	6,365	148	2.3
	Willard Parker Hosp.	3	113	5	4.4
	Kopetzky	5	273	28	10.2
Rochester	St. Mary's Hosp.	7¼	86	2	2.3
	Genesee Hosp.	12¼	484	0	0
	Strong Mem. and Municipal Hosps.	7	568	15	2.6
Syracuse	Britten	3	15	1	6.7
	Rubenstein	?	Ca. 75	3	4
Yonkers	Smith	?	646	12	1.9

TABLE 1.—*Incidence of Thrombosis of the Lateral Sinus in Mastoiditis—Continued*

Location	Source	Period Covered by Report, Years	Cases of Mastoiditis	Cases of Sinus Thrombosis	Incidence of Sinus Thrombosis, %
North Carolina					
Charlotte	Charlotte Eye, Ear and Throat Hosp.	5	219	19	8.7
Durham	Duke University Hosp.	3	50	0	0
Statesville	Davis Hosp.	?	137	3	2.2
Ohio					
Alliance	King	5	90	0	0
Canton	Hoffman	5	60	1	1.7
Cincinnati	City Hosp.	5	145	3	2.1
	Bryant	3½	40	0	0
	Mithoefer	10	Ca. 300	1	Ca. 0.3
	Vail	11	Ca. 100	6	Ca. 6
Cleveland	City Hosp.	5	313	3	1
	Lakeside Hosp.	6	720	20	2.8
	St. Alexis Hosp.	5	151	2	1.3
	St. Luke's Hosp.	1	127	5	3.9
Columbus	Beatty and Harris (2 hosps.)	6	154	15 (12 patients)	9.7
Dayton	Blackburn	15	Ca. 200	2	1
Youngstown	St. Elizabeth Hosp.	6	343	8	2.3
	Youngstown Hosp.	7	328	13	4
Oklahoma					
Oklahoma City	University Hosp.	7	140	3	2.1
Oregon					
Eugene	Norton	6	20	0	0
Portland	Good Samaritan Hosp.	2	?	2	?
	Doernbecher Child. Hosp.	1	80	0	0
	Fenton	1	51	1	2
Salem	Baum	6	54	2	3.7
Pennsylvania					
Abington	Abington Mem. Hosp.	6	?	3	?
Allentown	Allentown Hosp.	3	151	1	0.7
Chester	Chester Hosp.	2	22	0	0
Philadelphia	Bryn Mawr Hosp.	3	263	1	0.4
	Germantown Hosp.	5	448	3	0.7
	Frankford Hosp.	1	55	1	1.8
	Philadelphia Gen. Hosp.	8	167	2	1.2
	Presbyterian Hosp.	5	125	6	4.8
	Graduate Hosp.	11	644	9	1.4
	Woman's Hosp.	2	22	0	0
Pittsburgh	Allegheny Hosp.	12	955	8	0.8
	Eye and Ear Hosp.	10	844	30	3.6
	Guerinot	25	500	5	1
Reading	Reading Gen. Hosp.	3	107	5	4.7
Rhode Island					
Providence	Rhode Island Hosp.	?	?	?	2
South Carolina					
Greenville	Jervey	9	Ca. 100	0	0
South Dakota					
Hot Springs	Hot Springs Clinic	3	78	3	3.7
Pierre	Robbins	?	123	7	5.7
Tennessee					
Chattanooga	Hackney	15	Ca. 300	7	Ca. 2.3
Memphis	Baptist Mem. Hosp.	3	153	1	0.7
	Memphis Gen. Hosp.	5	?	3	?
	John Shea Clinic	1	32	1	3.1
Nashville	Kennon	?	131	13	9.9
Texas					
Dallas	Jones Eye, Ear, Nose and Throat Hosp.	5	158	6	3.8
	Baylor Hosp.	16	231	2	0.9
	St. Paul's Hosp.	?	129	2	1.6
El Paso	City-County Hosp.	No records of thrombosis of lateral sinus			
Fort Worth	Warwick	No records of thrombosis of lateral sinus			
	Rathberger	3	17	1	5.9
Galveston	John Sealy Hosp.	12	67	2	3
Houston		10	Ca. 50	1	2
Lubbock	Lubbock San. and Clinic	4½	170	2	1.2
San Angelo	Windham	10	Ca. 250	0	0
Vermont					
Barre	Jarvis	No records of thrombosis of lateral sinus			
Rutland	Seeley and Marshall	7	160	5	3.1

TABLE 1.—*Incidence of Thrombosis of the Lateral Sinus in Mastoiditis—Continued*

Location	Source	Period Covered by Report, Years	Cases of Mastoiditis	Cases of Sinus Thrombosis	Incidence of Sinus Thrombosis, %
Virginia					
Charlottesville	University Hosp.	7	319	18	5.6
Richmond	Med. Coll. of Virginia Hosp.	3	43	3	7.3
	Stuart Circle Hosp.	6	54	0	0
Roanoke	Gill Mem. Eye, Ear and Throat Hosp.	?	?	?	1
Washington					
Seattle	Seattle Eye, Ear and Throat Hosp.	?	487	26	5.3
Spokane	Deaconess Hosp.	3	173	4	2.3
Tacoma	Howe	8½	198	5	2.5
West Virginia					
Bluefield	Blaydes	10	432	14	3.2
Fairmont	Johnson	10	164	8	4.9
	Hall and Thomas	5	400	20	5
Wheeling	Zuback	12	112	2	1.8
	Wheeling Clinic	8	125	3	2.4
Wisconsin					
Fond du Lac	Folsom	?	126	2	1.6
La Crosse	Smith	5	200	1	0.5
Madison	Davis	13	105	3	2.9
Milwaukee	Milwaukee Hosp.	7½	336	5	1.5
Superior	Hovde	20	Ca. 800	3	0.4
Hawaii					
Honolulu	Pinkerton	5	65	4	6.2
	Poole	2	23	3	13
	CANADA				
Manitoba					
Winnipeg	Winnipeg Gen. Hosp.	3	329	13	4
British Columbia					
Vancouver	Vancouver Gen. Hosp.	5	675	21	3.1
Ontario					
Hamilton	Hamilton Gen. Hosp.	8	376	2	0.5
Kingston	Kingston Gen. Hosp.	2	34	3	8.8
Ottawa	Ottawa Civic Hosp.	10	376	10	2.7
Quebec					
Montreal	Royal Victoria Hosp	10	900	25	2.8
Nova Scotia					
Halifax	Victoria Gen. Hosp.	10	223	1	0.45
Yarmouth	Phinney	8	50	0	0

Only nineteen responses to this questionnaire indicated an incidence of thrombosis of the lateral sinus higher than 6 per cent. Seven of these represented small series, of less than 100 cases of mastoiditis; hence the percentages are not particularly important. It is interesting, though, and perhaps significant, that among the remaining twelve who reported high incidences were some outstanding specialists and authorities; for instance, Tobey, of Boston, who reported thrombosis of the lateral sinus in 7.9 per cent of a series of private patients; Kopetzky, of New York, who reported an incidence of 10.2 per cent, and the Mayo Clinic, at which the incidence was 9.4 per cent in a reported series. This may have been due to the high proportion of referred work, consisting of cases of the most serious mastoid disease. Other reports of a high incidence among groups consisting of referred cases came from the Charlotte (N. C.) Eye, Ear and Throat Hospital, 8.7 per cent; Vail, of Cincinnati, approximately 6 per cent, and Spratt, of Minneapolis, 8.4 per cent. The figures of Coates, Ersner and Persky furnished

a notable exception to this rule. They reported an incidence of only 1.4 per cent in nearly 1,000 cases of mastoiditis.

Three hospitals in the South (Lexington, Ky.; Louisville, Ky., and Nashville, Tenn.) showed incidences of 5.9, 6.2 and 9.9 per cent, respectively. As Kennon explained in commenting on the last figure, a large proportion of the patients in these institutions were Negroes and other poor folk who sought medical attention only as a last resort, when the ears were in the worst possible condition.

A great many of those who replied to this questionnaire remarked about the effect of climate on the incidence of thrombosis of the lateral sinus and of mastoiditis. In nearly all the letters from physicians in the southern states and many from those in California it was remarked that the climate lessened the virulence of otitic infection and hence affected the incidence of thrombosis of the lateral sinus. A few physicians in states such as Arizona, Texas, Georgia and Florida reported that they had never observed a case of thrombosis of the lateral sinus, and many reported only 1 or 2 cases in many years of practice.

In view of this opinion regarding the effect of climate on the incidence of thrombosis of the lateral sinus, it is interesting to see what the figures from certain places in various sections of the country actually show. In order that the comparisons may be as fair as possible, only series from hospitals are included in these tabulations, and in each instance the number of cases is greater than 200 in order that the incidence in percentage may be fairly reliable.

On the west coast, two hospitals in Seattle and Spokane, Wash., had 660 mastoidectomies, with 30 cases of thrombosis of the lateral sinus, an incidence of 4.5 per cent; four hospitals in San Francisco had 1,758 mastoid operations, with 32 cases of thrombosis of the lateral sinus, an incidence of 1.8 per cent; four hospitals in the Los Angeles area reported a total of 1,816 mastoid operations, with 70 cases of thrombosis of the lateral sinus, an incidence of 3.9 per cent.

In the South, three hospitals in Dallas, Texas, showed 518 cases of mastoiditis, with 10 instances of thrombosis of the lateral sinus, an incidence of 1.9 per cent. Three hospitals in North Carolina showed a total of 406 cases of mastoid disease with 22 cases of thrombosis of the lateral sinus (5.4 per cent). It should be noted, however, that one of these was an otolaryngologic hospital, doing largely referred work in otolaryngology.

In the Middle West, three hospitals in St. Louis showed 1,502 cases of mastoid disease with 55, or 3.7 per cent, cases of thrombosis of the lateral sinus. Four hospitals in Chicago, with 953 mastoid operations, had 24 cases of thrombosis of the lateral sinus, or 2.5 per cent. Three hospitals in Detroit had 2,244 cases of mastoiditis, with 39, or 1.7 per

cent, cases of thrombosis of the lateral sinus. There were 1,654 mastoidectomies in five hospitals in Cleveland and Youngstown, Ohio, with 38 cases of thrombosis of the sigmoid sinus, or 2.3 per cent. Two hospitals in Pittsburgh had 1,839 cases in which the mastoid was operated on, in 38 of which thrombosis of the lateral sinus occurred, an incidence of 2.1 per cent. Five hospitals in Minneapolis and Duluth, Minn., had a total of 599 cases of mastoid disease, with 4 instances of thrombosis of the lateral sinus, an incidence of 0.67 per cent.

On the east coast, two hospitals in Boston showed a total of 6,837 cases of mastoid disease, with 219 of thrombosis of the lateral sinus, or an incidence of 3.1 per cent. The records of four hospitals in New York yielded a total of 14,415 mastoid operations, with 335 cases of thrombosis of the lateral sinus, an incidence of 2.3 per cent. Seven hospitals in Philadelphia showed a total of 1,724 cases of mastoid disease with 22, or 1.2 per cent, cases of thrombosis of the lateral sinus.

In Canada, three hospitals in Ontario showed 786 cases of mastoid disease, with 15, or 1.9 per cent, cases of thrombosis of the lateral sinus. One hospital in Montreal sent records of 900 cases of mastoid disease, with 25 cases of thrombosis of the lateral sinus, or an incidence of 2.8 per cent. Two hospitals in Nova Scotia had records of 273 cases of mastoiditis, with only 1 instance of thrombosis, or an incidence of 0.4 per cent.

Of course, from the naked data, it is impossible to say just what influence climate had. It is interesting, nevertheless, that the two lowest rates of incidence were in cold districts, namely, Minnesota and Nova Scotia, and that the highest incidence, 5.4 per cent, was from a district (North Carolina) with an extremely moderate climate. This makes one question somewhat the significance of a warm climate in the incidence of thrombosis in mastoid disease, although apparently it does influence the number of cases of mastoid disease.

So far as is known, this series of 59,850 cases of mastoid disease is larger than any that has been analyzed in the literature, and the incidence of thrombosis is somewhat lower than that in most of the recorded series. Gill [1] of Roanoke, Va., said that the incidence of sinus thrombosis is between 5 and 10 per cent for both acute and chronic mastoiditis. Warren [2] stated that the incidence of thrombosis in otitis media is 3 to 5 per cent. Hamrick [3] gave the percentage as between

1. Gill, E. G.: Sinus Thrombosis and Thrombophlebitis Complicating Mastoiditis: Case Reports, South. M. J. **27**:718-727 (Aug.) 1934.

2. Warren, W. C., Jr.: Intracranial Complications in Purulent Otitis Media, Arch. Surg. **18**:1552-1561 (April) 1929.

3. Hamrick, D. W.: Lateral Sinus Thrombosis: Review of Literature and Report of Four Cases, New Orleans M. & S. J. **82**:130-135 (Sept.) 1929.

5 and 6. Wanamaker [4] reported 26 cases in 487 in which the mastoid was operated on in Seattle, an incidence of over 5 per cent. In Robbins' [5] series of 123 cases of mastoiditis, the incidence of thrombosis of the lateral sinus was 5.7 per cent. Meltzer,[6] in 4,961 cases at the Massachusetts Eye and Ear Infirmary, reported an incidence of thrombosis of the sigmoid sinus of 3.2 per cent. Smith [7] estimated the incidence as slightly over 2 per cent. Greenfield [8] and Coates, Ersner and Persky [9] gave the lowest figures in the recent literature, with incidences of 1.7 per cent in 1,257 cases of mastoiditis and 1.4 per cent in 969 cases of mastoiditis, respectively.

BILATERAL THROMBOSIS

The occurrence of bilateral infection of the lateral sinuses is apparently rare. Hastings [10] reported a case in which both jugular veins were ligated for bilateral thrombosis of the lateral sinuses, with a favorable outcome, and pointed out that bilateral ligation is possible, despite earlier warnings against it. He found 5 cases in the literature in which both jugular veins had been ligated during operation for a cancerous or tuberculous condition. Six cases had also been reported in which recovery took place after resection of one jugular vein in connection with bilateral sinal disease of otitic origin. Another case of this type was reported by Ruttin.[11]

In view of the extreme rarity with which this finding is reported in the literature, it is interesting that Beatty and Harris, of Columbus, Ohio, in answering the questionnaire reported that they had had 3 cases of bilateral involvement in a series of 12 cases of thrombosis. Two patients died and 1 recovered. Another case in which there was bilateral involvement of the sinuses was reported from the Royal Victoria

4. Wanamaker, A. T.: Facts and Factors in Lateral Sinus Thrombosis, Proc. Pan-Pacific S. Conf., 1929, pp. 337-345.

5. Robbins, C. E.: Thrombophlebitis of the Lateral Sinus, Journal-Lancet **52**: 677-685 (Nov. 15) 1932.

6. Meltzer, P. E.: Treatment of Thrombosis of Lateral Sinus: Summary of Results Obtained During Twelve Years at Massachusetts Eye and Ear Infirmary, Arch. Otolaryng. **22**:131-142 (Aug.) 1935.

7. Smith, A. J.: Spontaneous Perforation of the Sigmoid Sinus Following Mastoid Operation, Arch. Otolaryng. **17**:43-48 (Jan.) 1933.

8. Greenfield, S. D.: Thrombophlebitis of the Lateral Sinus, Arch. Otolaryng. **25**:661-683 (June) 1937.

9. Coates, G. M.; Ersner, M. S., and Persky, A. H.: Lateral Sinus Thrombosis, with Review of Literature, Ann. Otol., Rhin. & Laryng. **43**:419-440 (June) 1934.

10. Hastings, H.: Bilateral Jugular Resection for Bilateral Sigmoid Sinus Thrombosis (Otitic), Arch. Otolaryng. **4**:58-68 (July) 1926.

11. Ruttin, cited by Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **6**:162-172 (Aug.) 1927.

Hospital in Montreal, Canada. Both jugular veins were ligated in the patient, a child, with favorable results.

MORTALITY

Ninety-two replies to the questionnaire furnish exact information concerning the number of deaths from thrombosis of the lateral sinus.

TABLE 2.—*Mortality from Thrombosis of the Lateral Sinus*

City	Cases of Thrombosis of Lateral Sinus	Deaths	Mortality, %
Birmingham, Ala.	3	0	0
Selma, Ala.	4	0	0
Phoenix, Ariz.	5	2	40
Little Rock, Ark.	8	1	12
San Francisco	8	2	25
Bridgeport, Conn.	3	3	100
New London, Conn.	1	1	100
Wilmington, Del.	5	0	0
Washington, D. C.	1	0	0
Jacksonville, Fla.	5	2	40
Miami Beach, Fla.	1	0	0
Atlanta, Ga.	3	1	33
Chicago	13	6	46
Evanston, Ill.	3	1	33
Quincy, Ill.	4	1	25
Hammond, Ind.	14	2	14
Louisville, Ky.	6	3	50
New Orleans	4	1	25
Baltimore	30	10	33
Boston	202	64	32
Worcester, Mass.	7	3	43
Worcester, Mass.	5	1	20
Ann Arbor, Mich.	10	6	60
Detroit	14	1	7
Detroit	15	4	27
Grand Rapids, Mich.	2	1	50
Grand Rapids, Mich.	4	3	75
Lansing, Mich.	4	0	0
Minneapolis	21	4	19
Duluth, Minn.	4	4	100
St. Paul	10	8	80
Kansas City, Mo.	3	0	0
St. Louis	52	19	37
St. Louis	70	3	4
Lincoln, Neb.	3	1	33
New York	12	4	33
New York	5	4	80
New York	28	10	36
Rochester, N. Y.	15	4	27
Charlotte, N. C.	19	5	26
Statesville, N. C.	3	2	67
Cincinnati	6	2	33
Cleveland	3	1	33
Cleveland	20	7	35
Cleveland	2	1	50
Cleveland	5	1	20
Columbus, Ohio	12	5	42
Dayton, Ohio	2	1	50
Youngstown, Ohio	13	5	38
Abington, Pa.	3	2	67
Philadelphia	1	1	100
Philadelphia	3	2	67
Philadelphia	9	4	44
Pittsburgh	8	4	50
Pittsburgh	30	8	27
Pittsburgh	5	2	40
Hot Springs, S. D.	3	1	33
Chattanooga, Tenn.	7	2	29
Memphis, Tenn.	3	2	67
Nashville, Tenn.	13	3	23
Fort Worth, Tex.	1	0	0
Galveston, Tex.	2	2	100
Lubbock, Tex.	2	1	50
Rutland, Vt.	5	2	40
Charlottesville, Va.	18	8	44
Seattle	26	8	31
Spokane, Wash.	4	1	25
Tacoma, Wash.	5	1	20
Bluefield, W. Va.	14	4	29
Fairmont, W. Va.	28	14	50
Madison, Wis.	3	1	33
Honolulu, Hawaii	4	2	50
Honolulu, Hawaii	3	1	33
CANADA			
Winnipeg, Man.	13	6	46
Vancouver, B. C.	21	?	?
Hamilton, Ont.	2	1	50
Kingston, Ont.	3	1	33
Montreal, Que.	25	7	28
Halifax, N. S.	1	1	100

These replies concerned 979 cases of thrombosis, with 305 deaths, a mortality of 31.2 per cent. In individual series the mortality ranged from 0 to 100 per cent, but most of the larger series showed a death rate between 30 and 45 per cent. A notable exception was found in the record of the Mayo Clinic, with a mortality, according to Lillie, of 9.8 per cent (table 2).

The mortality computed from these collected series is similar to the rate reported by various authorities in the literature. In the series of 184 cases from the Massachusetts Eye and Ear Infirmary, reported by

Boies,[12] the total mortality was 25 per cent. From this list 18 in which the patient was admitted in a hopeless condition were excluded. The mortality reported by Coates, Ersner and Persky [9] was 35.7 per cent in a series of 14 cases. They commented, however, that 3 of the patients were hopelessly ill on admission. If these cases were excluded, the mortality was reduced.

Meltzer,[6] in a report of the results obtained during twelve years at the Massachusetts Eye and Ear Infirmary, divided the cases into two groups. In the first group, covering the period from 1921 to 1926, there were 64 cases with 20 deaths, a mortality of 31.3 per cent. When 6 cases in which the condition was hopeless on admission were subtracted from this, the mortality was reduced to 24.1 per cent. In the second group, covering the period from 1926 to 1932, there were 97 cases of thrombosis of the lateral sinus with 25 deaths, a mortality of 25.8 per cent. If the cases in which the condition was hopeless were subtracted from this group, the mortality was 20 per cent.

In the series of 52 cases from the University Hospital, Ann Arbor, Mich., reported by Maxwell,[13] the mortality was 36.5 per cent, with no cases excluded. McCarthy,[14] of Cincinnati, reported 10 deaths in a series of 48 cases, a mortality of 20.8 per cent. Dill and Crowe,[15] in a series of 30 cases from Johns Hopkins Hospital, Baltimore, reported a mortality of 33 per cent.

A compilation of 1,045 cases reported by seventeen authors was presented by Haymann,[16] of Munich, Germany, and the combined mortality was 34 per cent. Wolf [17] cited the reports of Brunner, of Vienna, Germany, with 22 per cent mortality in 154 cases; of Eliasson, of Leningrad, U. S. S. R., with 36 per cent in 121 cases, and of Mygind, of Copenhagen, Denmark, with 36 per cent in 199 cases. Kopetzky [18]

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12. Boies, L. R.: Lateral Sinus Thrombosis: Review of One Hundred and Eighty-Four Cases with Special Reference to Postoperative Type, Ann. Otol., Rhin. & Laryng. **41**:227-237 (March) 1932.

13. Maxwell, J. H.: Sigmoid Sinus Thrombosis: Clinical Analysis, Arch. Otolaryng. **25**:184-189 (Feb.) 1937.

14. McCarthy, M. F.: Some Experiences in the Surgery of the Lateral Sinus and Internal Jugular Vein, with Review of Fifty Cases, J. Med. **16**:614-620 (Feb.) 1936.

15. Dill, J. L., and Crowe, S. J.: Thrombosis of the Sigmoid or Lateral Sinus: Report of Thirty Cases, Arch. Surg. **29**:705-722 (Nov.) 1934.

16. Haymann, cited by Wolf.[17]

17. Wolf, G. D.: Analysis and Report of Ten Consecutive Cases of Sinus Thrombosis with Recovery, Laryngoscope **45**:940-947 (Dec.) 1935.

18. Kopetzky, S. J.: (*a*) Acute and Chronic Otitis Media, Sinus Thrombosis and Petrosal Pyramid Suppuration, Arch. Otolaryng. **18**:344-368 (Sept.) 1933; (*b*) Acute and Chronic Otitis Media, Sinus Thrombosis and Suppuration of the Petrous Pyramid, ibid. **22**:336-357 (Sept.) 1935.

cited the report of Fraser, in the German literature, which showed 40 per cent mortality, and Frödung's figures from Sabbatsberg, Sweden, showing a mortality of 26.7 per cent from the acute form of the disease and of 31.3 per cent from the chronic form.

Several authors have reported mortality conspicuously lower than that usually recorded. Portmann,[19] who claimed to have had no deaths during the last eight years, attributed his cures to prompt surgical intervention on both the lateral sinus and the internal jugular vein. Wolf [17] reported recovery in 10 consecutive cases in 1935. Potts,[20] of Omaha, reported a mortality of 14 per cent in the 63 cases of the Gifford group. The mortality reported by Wanamaker [21] was 15 per cent in his own series, but a mortality of 30 per cent was reported by the Seattle aural surgeons for four years. Greenfield [8] reported 3 deaths in a series of 22 cases, a mortality of 13.5 per cent. Lillie,[22] in a series of 52 cases reported from the Mayo Clinic, recorded a mortality of 13.4 per cent. It will be recalled that the figure given in his correspondence was 9.8 per cent. This probably included a longer series than that in the published report.

In interpreting these mortality figures, several factors must be taken into consideration. As has already been pointed out, some of them might be altered if the cases in which the condition is hopeless were to be excluded from consideration. When it comes to eliminating these cases, however, what criteria are to be used in deciding in which cases the condition is hopeless on admission? Boies,[12] in analyzing the figures in his series, set an arbitrary time of development of the signs of thrombosis of the lateral sinus as the deciding factor. Some other instances, in which the patient was apparently in serious condition at admission but in which symptoms of thrombosis of the lateral sinus were delayed, were included in the report. This merely emphasizes the difficulties encountered in trying to exclude certain cases from consideration.

It would simplify the situation and make evaluation of the records much more accurate if the figures always reported mortality exactly as it occurs without any attempt to make the record in certain series look more favorable by exclusion of some cases. In the treatment of this condition the surgeon is always bound to encounter patients that

19. Portmann, cited by Kopetzky.[35a]

20. Potts, J. B.: Thrombosis of the Lateral Sinus, J. A. M. A. **98**:379-382 (Jan. 30) 1932.

21. Wanamaker, A. T.: Some Considerations and Suggestions in the Handling of Lateral Sinus Thrombosis, Laryngoscope **40**:787-798 (Nov.) 1930.

22. Lillie, cited by Wolf.[17]

should have been cared for at an earlier date, but these must be considered as well as those whose condition is more favorable in any attempt to improve the results of treatment generally.

Many authors have built up lofty arguments concerning the efficacy of this or that procedure or the influence of this or that factor on the mortality. Despite all this, it is interesting and perhaps significant that in the larger proportion of all series of cases reported, the mortality ranged from 30 to 40 per cent. In the series compiled in this survey, a similar result, 31.2 per cent, was obtained.

Most authorities agree that certain factors influence the mortality, such as the time of diagnosis and the care of mastoid infections, which is influenced largely by the economic status of the patient; the virulence of the particular organism causing the trouble, and the chronicity of the infection. Boies'[12] analysis showed that the incidence of thrombosis was higher in cases of acute suppuration but that the mortality was greater in cases of chronic disease.

Many, if not most, of the deaths in thrombosis of the cases of lateral sinus are caused by complications, such as meningitis or intracranial spread of the infection, pulmonary complications or distant metastatic lesions, particularly in the vital organs. The incidence of these complications in association with thrombosis of the lateral sinus is high. In Boies' series, it was 46 per cent.

If one surgical procedure has an advantage over another so far as the mortality is concerned, this fact does not emerge clearly from the statistics available at the present time. The factors previously enumerated seem to have far more bearing than does the type of surgical procedure used. This subject will be discussed more fully under the heading of treatment.

ANATOMY

It is beyond the scope of this paper to enter into any lengthy discussion of the anatomy, pathology, etiology or bacteriology of thrombosis of the lateral sinus. These phases of the subject have been well reviewed by Seydell.[23] However, a number of investigators recently have contributed important anatomic studies bearing on the clinical findings in cases of thrombosis of the lateral sinus.

Swift[24] has given an excellent account of the embryology and anatomy of the lateral sinuses in relation to the remainder of the sinal

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23. Seydell, E. M.: Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **41**:466-492 (June) 1932.

24. Swift, G. W.: The Transverse Sinus and Its Relation to Choked Disk, Arch. Ophth. **3**:47-68 (Jan.) 1930.

system and has discussed its anomalies. Woodhall [25] reported a study on the variations of the cranial venous sinuses in a series of anatomic dissections. He found right-sided predominance in 39 per cent and left-sided predominance in 13 per cent. He observed inadequate cross circulation at the torcular Herophili in from 10 to 15 per cent of the specimens. Gross occipital abnormalities were present in from 4 to 6 per cent, and one lateral sinus was absent in 4 per cent. Woodhall pointed out that these anatomic anomalies should be kept in mind in evaluation of the results of the spinal manometric test for thrombosis of the lateral sinus and cited the ways in which they could affect the results of this diagnostic procedure.

The studies of Edwards [26] also called attention to the anatomic anomalies and their relation to the interpretation of the Tobey-Ayer test. He studied the anatomic variations in 50 skulls. Forty-six (92 per cent) showed a difference in diameter of the two venous channels. In 25 the right side and in 21 the left side was the wider. The variation was greater when the wider side was the right. Hence, he concluded that the measurement of both sinuses must influence the results of the Tobey-Ayer test. On compression of the wider vein, the venous stasis and fluid pressure would be higher than on compression of the narrower sinus.

Coates, Ersner and Persky [9] called attention to the clinical importance of these anatomic variations in the lateral sinuses. They found that anatomically the right sinus is larger than the left. They stated that it is extremely important to study carefully the relative size of the two lateral sinuses and the jugular foramens, from the standpoint of both operative procedure and prognosis. They expressed the belief that often atypical symptoms may be due to anomalies in sinal structure.

The clinical importance of anatomic anomalies has also been stressed by Effler,[27] who reported a case of absence of the lateral sinus. In commenting on this case, Kopetzky [28] stated that he had made a similar observation in his own practice and that the possibility of such a finding must be kept in mind. Levy and Laff [29] described a case in which no sinus was found at operation for thrombosis of the lateral sinus.

25. Woodhall, B.: Variations of Cranial Venous Sinuses in the Region of Torcular Herophili (Validity of Queckenstedt Test in Diagnosis of Thrombosis of Lateral Sinus), Arch. Surg. **33**:297-314 (Aug.) 1936.

26. Edwards, cited by Kopetzky.[18a]

27. Effler, cited by Kopetzky.[28]

28. Koptezky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **14**:489-511 (Oct.) 1931.

29. Levy, R., and Laff, H. I.: Sigmoid Sinus Thrombosis: Report of a Case, California & West. Med. **37**:233-238 (Oct.) 1932.

Hilding [30] and Williams [31] also have reported cases of absence of the lateral sinus.

Ziegelman [32] made an interesting anatomic study, which has a bearing on the surgical management of thrombosis of the lateral sinus. In examination of the mastoid emissary vein in 30 specimens he found that it was absent in 25 per cent. In the specimens examined this vein had a variable point of entrance into the venous circulation and differed considerably in size and direction. He expressed the belief that invasion of this vein in cases of thrombosis of the lateral sinus may account for some of the high mortality in this condition. This point has been emphasized by Hybasek,[33] who reported 3 cases of thrombosis of the mastoid emissary vein among 17 of thrombosis of otitic origin. He stated that when sepsis occurs and no thrombus is found in the sinus or bulb the mastoid emissary vein should be inspected.

PATHOLOGY

Seydell [23] has furnished a good general review of the etiology, pathogenesis and pathology of thrombosis of the lateral sinus. He stated that the factors which determine whether ordinary otitis media may terminate in disease of the lateral sinus are the type and virulence of the infective organism, the number of organisms present, the general and local resistance of the patient and the anatomic characteristics of the mastoid, such as the type of pneumatization and the facilities for drainage. He discussed also the various theories regarding the formation of thrombi in the lateral sinuses.

Coates, Ersner and Persky [9] have provided a good general discussion on these points and have shown that the usual offending organism is Streptococcus haemolyticus. This finding is the usual one, according to most authorities on the subject, although occasionally some other organism is isolated. Dill and Crowe [15] found hemolytic streptococci in 15 cases, nonhemolytic streptococci in 2, Pneumococcus type III in 1 and Bacillus proteus in 1. Friesner [34] obtained positive blood cultures in 90 of 100 cases of thrombosis of the lateral sinus. In 85 the organism was a streptococcus, in 2 Pneumococcus type III, in 2 Proteus and

30. Hilding, A.: False Response to the Jugular Compression (Tobey-Ayer) Test Due to Anomaly of the Lateral Sinus, Arch. Otolaryng. **26**:143-145 (Aug.) 1937.

31. Williams, H. L., Jr.: Apparent Unilateral Absence of Sigmoid Sinus Noted at Operation, Arch. Otolaryng. **12**:339-341 (Sept.) 1930.

32. Ziegelman, E. F.: Surgical Importance of Mastoid Vein in Infected Lateral Sinus Thrombosis, Arch. Otolaryng. **18**:298-309 (Sept.) 1933.

33. Hybasek, cited by Kopetzky.[28]

34. Friesner, I.: Infections of the Middle Ear: Acute Systemic Infections from the Ear, Arch. Otolaryng. **14**:257-265 (Sept.) 1931.

in 1 Staphylococcus aureus. In my own experience, Pneumococcus type III has been second to the hemolytic streptococcus as the causative organism. Kopetzky [35] has cited 2 cases, reported by Fioretti and by Segar and Stoeffler, in which the causative organism was proved to be Streptococcus viridans and a staphylococcus, respectively.

CLASSIFICATION

Boies [12] made an interesting classification of cases of thrombosis of the lateral sinus, which he divided into three types: those in which the condition is present at the time of the mastoid operation, those in which it is not apparent then but latent and those in which it is truly postoperative. In only 19 of the 184 cases he analyzed could the condition be classified as postoperative. In all the others, he decided from the clinical evidence, it was either apparent or latent at the time of the mastoid operation. He found that the prognosis is more favorable in the cases in which the condition was postoperative, and that the mortality is lower in that group.

Other classifications of cases of thrombosis of the lateral sinus have been based on the type of thrombus or the site and extent of the area involved, such as the jugular bulb, the torcular Herophili or the mastoid emissary vein. Kopetzky [36] called attention to a type of phlebitis which must be distinguished from infected thrombosis of the lateral sinus, namely, infection and thrombosis of the smaller vessels in the mastoid area. He called this condition hemorrhagic mastoiditis, while Beck called it thrombotic mastoiditis. This classification is important clinically, because this condition is often accompanied with septic temperature and other signs pointing to thrombosis of the sinus which is not found at operation.

SYMPTOMATOLOGY AND DIAGNOSIS

The common symptoms of thrombosis of the lateral sinus have been discussed in detail by Seydell,[23] Robbins,[5] Meltzer,[6] Almour,[37] Coates, Ersner and Persky [9] and Dill and Crowe.[15]

The comment that Tobey [38] made years ago concerning symptomatology and diagnosis is still apropos. He said:

> The terms "typical" and "atypical," as applied to cases of lateral sinus thrombosis, should be discarded from the literature, since they are most misleading, not only to

35. Kopetzky, S. J.: (*a*) Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **16**:575-583 (Oct.) 1932; (*b*) footnote 28.

36. Kopetzky, S. J.: Acute and Chronic Otitis Media and Thrombosis, Arch. Otolaryng. **10**:302-313 (Sept.) 1929.

37. Almour, R.: Prevention and Minor Symptoms of Sinus Thrombosis, with Case Reports, Arch. Otolaryng. **6**:373-382 (Oct.) 1927.

38. Tobey, G. L.: Lateral Sinus Thrombosis, Tr. Am. Otol. Soc. **15**:160-181, 1919.

the beginner, but to the older otologists, who are not meeting with a large number of these cases. I feel justified in making the statement that the textbook so-called "typical case" is very rare, and the otologist must be taught that each case is a problem unto itself, and that the picture is rarely complete.

Coates, Ersner and Persky [9] called attention to the fact that so-called atypical symptoms may be due to anomalies in sinal structure, to involvement of the bulb or of the petrosal and other sinuses or to infection by organisms other than the hemolytic streptococcus.

If all infections of the lateral sinus were accompanied with chills, a marked rise in temperature and a positive blood culture, the diagnosis would be comparatively easy, but such is not the case. This so-called classic picture is by no means distinct and definite in every case. According to Tobey,[38] less than 50 per cent of patients exhibit definite chills, although a greater number may have some chilly sensations which are discovered only by asking direct questions. Of the series of cases reported from the Johns Hopkins Hospital Dill and Crowe [15] found that chills were present in just half, while in two thirds of them there was a typical septic temperature curve. They obtained positive blood cultures in 9 of 21 cases, in which culture was done.

Tobey [38] stressed four different types of temperature curve which may be present in cases of thrombosis of the lateral sinus. These include (1) the accepted picture of true sepsis, with sudden elevations and depressions, (2) an irregular temperature curve which at no time shows an elevation of more than 2 or 3 degrees, (3) an elevation of 2 to 3 degrees which does not remit to normal but shows slight remissions and elevations from 100 degrees as a basis and (4) no elevation in temperature at all. Tobey observed 11 instances of the last type in a series of 73 cases of proved thrombosis of the lateral sinus.

Another possibility that must be kept in mind in interpreting a septic temperature curve is that this may occur after mastoid operations without thrombosis of the lateral sinus. Beyer [39] and Lazera and Torres [40] have reported such cases. As Kopetzky [35a] has emphasized, in most instances the manifestation is probably due to hemorrhagic mastoiditis. Levy and Laff [29] reported a case of otitic sepsis in which the sigmoid sinus was absent. Since the patient recovered after the eradication of hemorrhagically inflamed mastoid, they concluded that the sepsis was due to osteothrombotic phlebitis of the small veins. Occasionally, too, if too much reliance is placed on the temperature curve, thrombosis of the lateral sinus may be diagnosed in cases of pneumonia. In the older literature the differential diagnosis of thrombosis of the lateral sinus

39. Beyer, T. E.: Otitic Pyemia Without Sinus Thrombosis, Laryngoscope **38**:785-789 (Dec.) 1928.

40. Lazera and Torres, cited by Kopetzky.[35a]

and typhoid fever was often discussed, but with the present rarity of the latter disease, the importance of this differentiation has diminished.

Whenever the temperature curve following mastoidectomy is suggestive of septicemia, the diagnosis of thrombosis of the lateral sinus should not be made until a thorough general examination, an examination of the urine, a blood count, blood culture and a spinal puncture with a Tobey-Ayer test have been done. In my experience, incipient erysipelas, pyelitis, acute nephritis and acute tonsillitis have been frequent causes of a sudden rise in temperature following aural surgery.

Thrombosis of the lateral sinus may be associated with latent mastoiditis, and in such instances the true diagnosis may be masked because of the apparently mild degree of otitic infection. Kemler [41] and Dixon [42] called attention to this association particularly.

Apropos of the presence of choked disk, Tobey [38] observed that it was present in only 10 to 11 per cent of cases. Friesner [34] found papilledema of some degree in 16 per cent of his series of 100 cases. It was present in 12 of 26 cases in Dill and Crowe's [15] series. Benedict [43] showed that choked disk may be associated with disease of the mastoid process without evidence of thrombosis of the lateral sinus. Hence, the finding of choked disk seems to be of little definite significance in the diagnosis of thrombosis of the lateral sinus. Benedict said:

> With the knowledge that choked disk may occur in surgical disease of the mastoid process, with or without thrombosis of the lateral sinuses, the only conclusion one can reach regarding the significance of choked disk is that some intracranial involvement has taken place, but that the nature of this involvement is not manifest.

Dill and Crowe and Benedict commented that the presence of choked disk does not necessarily add gravity to the prognosis, for complete recovery had occurred in a number of cases in which this symptom was present.

Tobey [38] found that the most constant objective symptom in his cases was edema or infiltration with tenderness on deep pressure over the area drained by the mastoid emissary vein. He mentioned that the headache, usually unilateral, is rarely described as a pain but as a dull pressure.

Almour [37] called attention to some of the minor symptoms of thrombosis of the lateral sinus and observed that the presence of this

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41. Kemler, J. I.: Unrecognized and Latent Mastoiditis with Lateral Sinus Thrombosis, Laryngoscope **42**:101-107 (Feb.) 1932.

42. Dixon, O. J.: Symposium on Otolaryngological Problems in Sepsis: New Conceptions in Management of Septic Thrombosis, Laryngoscope **44**:448-453 (June) 1934.

43. Benedict, W. L.: Cranial Sinus Thrombosis: Ophthalmologic Aspects, Surg., Gynec. & Obst. **52**:464-469 (Feb. 15) 1931.

condition may not necessarily give the typical picture of sepsis. Among the symptoms he noted were unilateral headache, persistent polymorphonucleosis, papilledema, an appearance of well-being and perhaps an overlooked initial chill.

There is some difference of opinion as to the diagnostic significance of the appearance of the sinal area at operation on the mastoid. Many authors have declared that the appearance of the sinal wall gives no indication of what may be within it. A contrary opinion was stated by Greenfield,[8] who expressed the belief that the pathologic picture of the sinal wall during operation on the mastoid area is of great diagnostic significance.

Kopetzky [28] commented on the significance of pulsation in the sinus when it is exposed. He stated that many otologists seem to have the erroneous opinion that the sinus pulsates normally. I can corroborate this for I have had numerous arguments with confrères on this very point. Kopetzky based his comment on an article by Valenti, who called attention to the clinical significance of pulsation of the sinus, which occurs only when the elasticity of the sinus has been pathologically altered, and then the pulsations are indicative of a thrombus within the vein. A thrombosed sinus may not pulsate, but when such is the case it is highly probable that the thrombus has undergone purulent disintegration.

As for the laboratory procedures which aid in the diagnosis of thrombosis of the lateral sinus, the one stressed most is the blood culture. Nearly all authors have emphasized the importance of the positive blood culture, although they have noted that it is not obtained in all cases of thrombosis of the lateral sinus, and that when other signs and symptoms point the way operation should not be delayed because blood culture is negative. Friesner [34] stated that he sets great store by the positive blood culture and reported a higher incidence of this manifestation than other workers. He expressed the opinion that its importance is often overlooked in the diagnosis of thrombosis of the lateral sinus. He reported 90 per cent positive cultures in a series of 100 cases. He said in some instances the thrombus may remain sterile at both ends and hence not produce a positive blood culture. As Fenton [44] pointed out, it is a matter of experience that blood cultures are more likely to be positive if the blood is obtained at the beginning of a chill.

The blood count is a routine procedure in cases of suspected thrombosis of the lateral sinus, as of all other types of infection, and all workers have reported a high polymorphonuclear count. Other examinations of the blood, such as the Schilling and the sedimentation test, are widely

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44. Fenton, R. A.: Some Observations on the Management of Infections of the Blood Stream from Mastoiditis, Arch. Otolaryng. **25**:618-622 (June) 1937.

used in the diagnosis of this condition. Coates, Ersner and Persky [9] stated that these tests are especially valuable.

The Tobey-Ayer test is the procedure most widely used to determine the side of the infected thrombus. Tobey,[45] in commenting on this test, which is a modification of the Queckenstedt test largely employed in Europe, mentioned all the dangers of and contraindications for its use. Almost all authors who have had experience with the test have expressed agreement that it is valuable within a limited sphere. Seydell [23] said that it is of help but not absolutely reliable. Coates, Ersner and Persky [9] stated that it has definite value, but must be used and interpreted in connection with other findings in making the diagnosis. Tunstall [46] reviewed the results of this diagnostic procedure and concluded that they were, on the whole, favorable but that the test is useless for crying children, that a mural thrombus gives negative findings and that the findings may be altered by the collateral circulation in a thrombus of long standing. In the series reviewed by Meltzer,[6] the Tobey-Ayer test was found to give correct results in 75 to 80 per cent of the cases in which it was employed.

Woodhall [25] warned that its results are likely to be influenced adversely by anomalous anatomic findings. Woodhall's comments gain weight with clinical reports, such as that of Hilding,[30] of a false response to the Tobey-Ayer test in a case in which one lateral sinus was absent. The reaction to the test indicated that the left lateral sinus was closed and that the right one was open. This proved at autopsy to be correct, but the closure on the left was due to a developmental anomaly and not to thrombosis. The right sinus was extremely large and wide open but contained a septic mural thrombus, the source of the infection. Operation was not performed because of the poor condition of the patient, but it would have been fruitless if it had been attempted on the left side. Kopetzky [47] also recorded an instance in which the Tobey-Ayer test give misleading information. The manometric pressure was equal on both sides in a case in which there was thrombosis on one side, proved by operation.

Two variations of the Tobey-Ayer test that have been proposed are worthy of mention. Dandy [48] suggested ventricular puncture in cases in

45. Tobey, G. L., Jr.: The Diagnosis of Lateral Sinus Infection, Atlantic M. J. **30**:759-762 (Sept.) 1927.

46. Tunstall, C.: Thrombosis of Sigmoid Sinus and Internal Jugular Vein with Special Reference to Tobey-Ayer Test, South. M. J. **23**:639-645 (July) 1930.

47. Kopetzky, S. J.: Observation on Diagnosis of Lateral Sinus Thrombosis Contradictory to Results of Tobey and Ayer, Arch. Otolaryng. **7**:532-533 (May) 1928.

48. Dandy, W. E.: Cerebral (Ventricular) Hydrodynamic Test for Lateral Sinus Thrombosis, Arch. Otolaryng. **19**:297-302 (March) 1934.

which ventriculograms are made, to save the patient another puncture of the spine. The principle of this examination is the same as that of the spinal manometric determination. Another variation of this principle has been proposed in the German literature by Fleischman.[49] This investigator said that if one hinders the free flow of blood on the healthy side by compressing the internal jugular vein there is immediately an engorgement of the veins in the optic disk on the diseased side. If there is no obstructing thrombus, no changes in the fundus are noted.

Experimental attempts at visualization of the cranial sinuses by roentgenography have been made in this country by Dixon [50] and in Europe by Frenckner,[51] but these methods have not been sufficiently developed to have any significance in clinical diagnosis.

In general, it may be said that the answers to the questionnaire contained little specific comment on the diagnosis of thrombosis of the lateral sinus. One or two contained comment as to the value of the Tobey-Ayer test, if positive, in demonstrating the involved side. In one there was comment that the presence of choked disk is of great significance and that repeated ocular examinations should be made for this finding. Brenneman, of Chicago, summed up the experience and opinion of most members of the medical profession succinctly when he said, "I think the diagnosis of sinus thrombosis is one of the most difficult in medicine to make with any assurance of accuracy, except by operation."

TREATMENT

It is in the matter of treatment of thrombosis of the lateral sinus that the most divergence of opinion exists. In earlier years there was some controversy over whether the jugular vein should be ligated or resected, but lately there has been a new school of thought which condemns operation on the jugular vein and advocates that surgical procedures should be limited to drainage of the sinus. Protagonists of each opinion substantiate their arguments with records which appear convincing, and yet, with few exceptions, the mortality remains uniform, regardless of the type of treatment used.

In view of the discussion which waxes warm in the otologic literature today, it is interesting to see what methods are actually being used in practice, as revealed by the questionnaire. Only forty-three replies furnished definite information regarding operation or the type performed. From these answers, exposure of the sinus with ligation of

49. Fleischman, cited by Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Suppuration of the Petrous Pyramid, Arch. Otolaryng. **20**:396-421 (Sept.) 1934.

50. Dixon, O. J.: Research Studies in Visualization of the Vascular Supply of the Head and Neck, Tr. Am. Acad. Ophth. **39**:351-366, 1934.

51. Frenckner, cited by Kopetzky.[18b]

the jugular vein seemed to be the method preponderantly employed. Twenty-one replies stated that the operation performed was exposure with ligation; seven mentioned both resection and ligation, four, exposure with resection, and two, exposure of the sinus with no ligation; three stated that no operation was performed; six said that operations were performed but did not mention the type.

The only replies received which recalled the older controversy regarding ligation as opposed to resection came from the Manhattan Eye and Ear Hospital and the New York Eye and Ear Infirmary. From the former, 170 cases of thrombosis of the lateral sinus were reported, in which 168 resections and 2 ligations were performed. From the New York Eye and Ear Infirmary, 148 cases of thrombosis of the lateral sinus were reported, with 118 ligations and 30 resections. No comments were included with these records, but the reversed ratios of the two types of operation on the jugular vein in these two metropolitan institutions are interesting.

In the literature, a number of authors have stated emphatically that operation on the jugular vein should be performed in all cases. Among these are Portmann,[19] Feuz,[52] Warren,[2] Hartman,[53] Parker,[54] Dunn and Cowan,[55] Seydell,[23] Gill,[1] Greenfield[8] and Friesner.[34] Among those who have stated that operation on the jugular vein is seldom or practically never indicated are Meltzer,[6] who commented that the majority of the undesirable complications occur after ligation; Dixon,[56] who expressed the belief that the jugular vein should never be touched in these cases; Rott;[57] Richards;[58] Mygind,[59] of Copenhagen, Denmark, and Robbins,[5] who expressed the belief that ligation does not influence the outcome but advised immediate opening of the sinus.

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52. Feuz, cited by Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Suppuration of the Petrous Pyramid, Arch. Otolaryng. **24**:505-526 (Oct.) 1936.

53. Hartman, E. C.: Diagnosis and Management of Lateral Sinus Thrombosis, West Virginia M. J. **26**:541-543 (Sept.) 1930.

54. Parker, R. H.: Lateral Sinus Thrombosis Complicating Mastoiditis, J. Iowa M. Soc. **23**:136-140 (March) 1933.

55. Dunn, A. G., and Cowan, W. F.: Lateral Sinus Thrombosis Complicating Acute Mastoiditis, Wisconsin M. J. **33**:836-838 (Nov.) 1934.

56. Dixon, O. J.: (*a*) Non-Ligation of Internal Jugular Vein in Treatment of Sigmoid Sinus Thrombophlebitis, Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:142-152, 1932; (*b*) Departure in Management of Thrombophlebitis of Sigmoid Sinus, Based on Experimental Studies on the Dog, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1932, pp. 138-149.

57. Rott, O. M.: Why Ligate the Jugular Vein in Cases of Lateral Sinus Thrombosis? Arch. Otolaryng. **14**:272-281 (Sept.) 1931.

58. Richards, L.: Pros and Cons of Jugular Ligation in Lateral Sinus Thrombosis, Kentucky M. J. **33**:351-354 (Aug.) 1935.

59. Mygind, S. H.: Treatment of Otogenic Sepsis and Sinus Thrombosis, Acta oto-laryng. **16**:474-486, 1931.

Ersner and Myers [60] have summed up the case against ligation most recently, and their conclusions are typical of those of the members of this school of thought. They argued that ligation is superfluous, since the extension of the infection from the lateral sinus is retrograde rather than downward, and they cited the physiologic studies of Nielsen and Courville,[61] Dixon [62] and Stone and Berger [63] to support this view. They said, "A thrombus is therefore a physiologic protective mechanism having a pathologic basis for its formation and is nature's response . . . to a potential danger, and it is an important protective element in the line of defense." These authors stressed the medical treatment as the most important factor in the management of the condition. They advocated transfusions of whole blood, immunotransfusions, administration of specific serums, forced feeding, the giving of tonics and ultraviolet irradiation, the use of such drugs as sulfanilamide and quinine and in some instances the production of a specific abscess by the injection of an irritating substance, such as turpentine. The only surgical intervention they said was justifiable was promotion of drainage.

They concluded that thrombosis is a protective process, that ligation of the internal jugular vein is not a panacea in the treatment of infection of the lateral sinus, that ligation of the internal jugular vein does not prevent metastasis, embolism or septicemia, that the treatment of infection of the lateral sinus is primarily medical and partially surgical, that the surgical principle is the institution of drainage and the avoidance of overmanipulation and lastly that thrombectomy is not indicated when the thrombus is firmly fixed.

Between these opposing camps, that is, the one advocating ligation in all cases and that advocating no ligation or resection of the jugular vein at all, there is a middle group which has stated that treatment must be individualized and that no generalizations can be made to apply in all instances. In this group are Almour,[64] Dill and Crowe,[15] Maxwell,[13]

60. Ersner, M. S., and Myers, D.: (*a*) Outstanding Signs and Symptoms: Merits and Demerits of Internal Jugular Ligation in the Treatment of Lateral Sinus Thrombosis, Pennsylvania M. J. **39**:579-585 (May) 1936; (*b*) Treatment of Thrombosis of the Lateral Sinus Without Ligation of the Internal Jugular Vein, J. A. M. A. **109**:919-923 (Sept. 18) 1937.

61. Nielsen, J. M., and Courville, C. B.: Intracranial Complications of Otogenous Thrombosis of the Lateral Sinus, Ann. Otol., Rhin. & Laryng. **46**:13-38 (March) 1937.

62. Dixon, O. J.: Vascular Repair: Use of Viable Muscle in Operations on the Sigmoid Sinus; Experimental Studies, J. A. M. A. **103**:1129-1135 (Oct. 13) 1934; footnote 56 *b*.

63. Stone, F. E., and Berger, M. D.: Retrograde Sinus Thrombosis Complicating Primary Thrombosis of the Jugular Vein, Arch. Otolaryng. **24**:141-158 (Aug.) 1936.

64. Almour, R.: Discussion of the Basis for the Selection of the Type of Procedure in Sinus Thrombosis, Laryngoscope **44**:454-464 (June) 1934.

who has expressed belief in operation on the jugular vein in most cases, Krepuska, Kopetzky and Mackenzie.[65] My own opinion agrees essentially with this last view. Avoidance of ligation in all cases seems to me dangerous unless one can be sure that the thrombus does not extend into the jugular bulb.

A minor controversy in the treatment of thrombosis of the lateral sinus centers about the efficacy of transfusions. Lillie [66] recommended blood transfusions in cases in which the temperature remains persistently septic and in which there is increasing anemia. Kopetzky [36] expressed agreement that blood transfusions should be given for anemia but disagreement with Lillie's conclusions regarding the effect of transfusion on septic temperature. He said that a persisting septic temperature curve is an indication that the focus of infection has not been relieved, and that repeated transfusions dilute the antibody reaction of the patient's blood and are a hindrance to speedy recovery. Friesner,[34] who had wide experience with septicemia, stated: "I have seen no evidence that would make me believe that transfusions of any character contribute except indirectly to the cure of the condition. They contribute no more than food or any other supportive measure does." On the other hand, Fenton [44] recently expressed belief in the efficacy of transfusions in the treatment of infections of the blood stream and Ersner and Myers made the statement that "small and frequently repeated transfusions act to stimulate the formation of antibodies."

Although there is this disagreement regarding the effect of transfusions, the evidence from the correspondence and from published material indicates that practically all otologists employ them in the treatment of septicemia accompanying infection of the lateral sinus.

In view of the widespread publicity which sulfanilamide has received in the recent literature and the hope that it has aroused for the successful treatment of various types of infection, some mention of this drug should be made. Ersner and Myers [60b] recommended its use in the medical management of the condition, and Fenton [44] has commented especially on this drug in connection with cases of thrombosis of the lateral sinus. He stated that because of its peculiar action in augmenting the phagocytic activity of the leukocytes its use offers considerable hope in the management of infections of the blood stream and may accomplish what the earlier attempts to inject antiseptics into the blood stream failed to do. Kolmer [67] recommended sulfanilamide administered

65. Mackenzie, G. W.: Sinus Thrombosis, Hahneman. Monthly **67**:438-441 (June) 1932.

66. Lillie, cited by Kopetzky.[36]

67. Kolmer, J. A.: Etiology, Prophylaxis and Treatment of Surgical Septicemia: A Discussion of the Principles Involved, Arch. Otolaryng. **26**:59-65 (July) 1937.

orally and prontosil [68] intravenously or intramuscularly for septicemia due to the hemolytic streptococcus.

Most physicians who have used sulfanilamide have had occasion to marvel at the results obtained in certain instances, but I am sure its actions are not comprehended fully. As usually occurs when any new therapeutic agent gains widespread use, the most recent literature is flooded with reports of various types of undesirable reactions following the use of sulfanilamide. These should not dampen enthusiasm but should make physicians ever alert and careful in administering the drug. It is only by the experiences of many physicians, both favorable and unfavorable, that its limitations and value can be accurately defined.

In my own experience with sulfanilamide I have observed in several instances a fall in temperature and an apparent remission of symptoms, followed by an exacerbation after the drug was discontinued. It may be that the action of sulfanilamide is such that the improvement is more apparent than real and that its effect may mask the true condition of the patient. At any rate, I feel that this agent must be administered cautiously and that the condition of any patient receiving it must be watched assiduously.

Blood counts and determinations of hemoglobin should be made daily, but it is not necessary to discontinue or reduce the dose of the drug unless the loss in hemoglobin or reduction of white blood cells is rapid. The milder toxic effects can be combated by the use of iron by mouth and by blood transfusions at frequent intervals.

The amount of the drug to be given depends on the severity of the infection and the tolerance of the patient; however, if given at all, the dose should equal 1 grain per pound (0.14 Gm. per kilogram) of body weight each twenty-four hours. A 150 pound (68 Kg.) patient should receive 150 grains (9.72 Gm.) per day, and as the condition improves the amount should be reduced. In the treatment of mastoid disease with symptoms of infection of the blood stream it is well to continue the use of the drug for at least two weeks after the temperature is normal. To prevent acidosis, sodium bicarbonate is given in amounts equal to the dose of sulfanilamide. The use of saline cathartics is contraindicated during the time sulfanilamide is being used.

Whatever the discussion regarding treatment, the fact remains that treatment of thrombosis of the lateral sinus is still fixed largely in the domain of the surgeon, for even authors who denounce operations on the internal jugular vein agree that in most cases the drainage of the sinus must be promoted by surgical means. There is rather general agreement also that operation should be performed as early as possible and hence that early diagnosis is one of the most important factors in the successful management of the condition.

68. The disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′, 6′-disulfonic acid.

PROGNOSIS

Despite the variety of treatment advocated for thrombosis of the lateral sinus, the outcome in all large series of cases remains much the same. Since in approximately one third of the cases the condition results fatally, the prognosis must always be guarded. Various factors such as the age of the patient, the time of appearance of the infection of the blood stream in relation to the mastoiditis and the general condition of the patient seem to be the most important in determining the outcome. The prognosis is more grave for older patients, especially those more than 40, and for those who have had sepsis for a considerable time before the operation. Maxwell [13] called attention to the fact that the prognosis is worse when the thrombus extends into the torcular Herophili or the internal jugular vein. Meltzer [6] stated that the outcome depends on the battle for supremacy between the infecting organism and the patient's power of resistance.

Death usually results from some complication, such as spread of the infection into the brain, with meningitis or abscess of the brain, and metastasis to other organs. Boies [12] stated that pulmonary complications were the most common in his series, with cerebral complications and orthopedic metastases next in order.

Nielsen and Courville [61] furnished a good review of intracranial complications of otogenous thrombosis of the lateral sinus, which they said are usually due to venous obstruction or to retrograde extension of infection into the afferent vessels and thence into the brain. The anatomic arrangement of the intracranial venous system accounts largely for the distribution and character of many of these lesions. They found that in most cases of fatal otogenous thrombosis some other intracranial lesion was seen at autopsy. Such a lesion may be coincidental, developing as a result of extension along some other path from the middle ear, or consequential, due directly to the thrombosis in the lateral sinus.

COMMENT AND SUMMARY

A questionnaire addressed to approximately a thousand physicians and hospitals brought three hundred and forty-three replies. From this correspondence a collective series of 59,850 cases of mastoid disease was compiled, with 1,556 cases of thrombosis of the lateral sinus. These figures yield an incidence of 2.6 per cent for thrombosis of the lateral sinus in mastoid disease.

Only ninety-two replies contained specific information regarding mortality. From these, a series of 979 cases of thrombosis was compiled, with 305 deaths, a mortality of 31.2 per cent.

In general, the incidence of thrombosis seems to be higher in children's hospitals and charity hospitals and in private practice largely among referred patients. There is no evidence from the statistics here assembled that climate has any influence on the incidence of thrombosis of the lateral sinus in mastoiditis, although it may affect the incidence of mastoid infection itself.

There is no evidence from either the correspondence or a survey of recent literature that the type of treatment influences the mortality. This seems to be affected more by the age of the patient, the virulence of the infection and the presence or absence of complications.

Despite the growing number of authors in the literature who oppose operative intervention on the jugular vein in the treatment of thrombosis of the lateral sinus, the survey shows that exposure of the sinus with ligation of the jugular vein is still the method of choice of a preponderant number of otologists. This surgical procedure is accompanied with transfusions and the administration of specific serums, tonics and drugs, such as sulfanilamide.

There are evident disagreement about the wisdom of attacking the jugular vein and the efficacy and effect of transfusions and a great deal of discussion about the role that sulfanilamide is to play in the treatment of this condition. These controversies and discussions emphasize that much is yet to be learned about this serious complication of mastoid disease.

Since the experience of the individual otologist with thrombosis of the lateral sinus is necessarily limited by the rarity of the conditions, the only way by which additional knowledge and better management are to be acquired is by the collective experience of the members of the medical profession.

This fact leads to a plea to the individual physician and surgeon to make careful observations and complete and accurate records. This survey furnishes adequate proof of the need for such records. Almost half of the persons who replied to the questionnaire could not give exact information, either because they had not kept records or because their records were not in such shape that the information was easily available.

The importance of all possible knowledge of a condition in which the mortality is approximately one third is obvious. The individual otologist can hope for improved results only if the collective experience and wisdom of the members of the profession is available to him.

References to thirty-five medical publications, which have been reviewed and have had a part in the formation of the opinions and comments expressed, though they have not been alluded to by direct citation, are included in the author's reprints.

The names and addresses of three hundred and forty-three contributors and correspondents will also appear in the reprints.

Case Reports

TUBERCULOSIS OF THE MAXILLARY SINUS

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Chronic suppuration of the paranasal sinuses is commonplace in the rhinologist's practice and generally responds well to treatment. Occasionally cases are encountered in which the infection is resistant to treatment, and one is hard pressed to explain why it persists. Despite this chronicity, disease of the paranasal sinuses and tuberculosis are seldom thought of in the same category. When bacteriologic studies are made of the nasal discharge or washings, the possible presence of acid-fast organisms is ignored. It may be because of this oversight that tuberculosis of the paranasal sinuses is occasionally overlooked. That the condition is rare cannot be denied. Herein is reported a case in which chronic suppurative maxillary sinusitis failed to respond to radical treatment and was finally proved to be tuberculous.

REPORT OF A CASE

A 26 year old Puerto Rican woman was admitted to the hospital on March 5, 1937. She was complaining of pain in the right cheek and in the lower part of the abdomen.

The pain in the lower part of the abdomen was first noticed in November 1935, after the patient had lifted a heavy object while she was employed as a factory worker. She sought treatment, but the pain continued unchanged.

In February 1936 she was admitted to a hospital for the treatment of lobar pneumonia. After recovery she was transferred to the otolaryngologic service because of the pain in the right cheek. Radical operation for chronic suppurative maxillary sinusitis was performed. After the operation a fistulous tract extended from the right antrum to the right gingivolabial sulcus.

The patient's abdominal pain persisted after she was discharged from the hospital. A gradually enlarging mass was noted in her right loin on successive examinations. She reentered the hospital, where the mass was aspirated on two occasions. The aspirated fluid contained tubercle bacilli, demonstrated by culture and by inoculation of a guinea pig.

On March 5, 1937, the patient was admitted to the Sea View Hospital.

At the time of admission examination showed her to be well developed but poorly nourished and chronically ill looking. The right cheek, the periorbital tissues and the right submaxillary areas were swollen. Tenderness was discernible on palpation over the right antrum. Several large, tender, discrete and firm glands were felt in the right cervical and submaxillary region.

From the Otolaryngological Service of Dr. M. C. Myerson, Sea View Hospital.

Above the right upper molar teeth a fistulous tract opened, from which exuded creamy white pus.

In the inferior meatus anteriorly on the right side there was a large opening into the antrum.

In the right lower quadrant of the abdomen there was a firm, nonmovable, nontender mass which measured 4 by 8 by 12 cm. This seemed to be the extension of a firm nontender indurated mass immediately to the right of the lumbar segment of the spine.

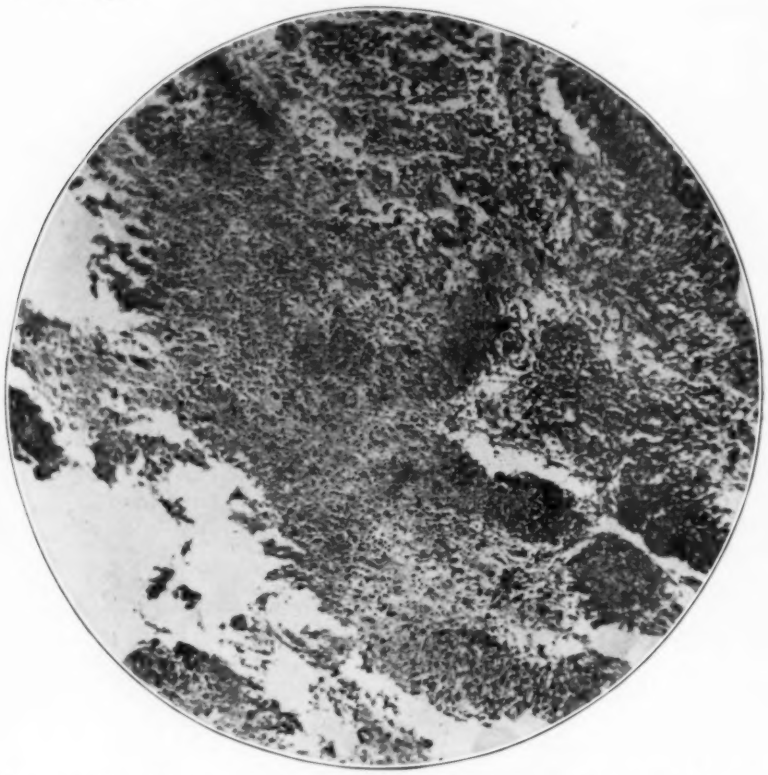

Fig. 1.—Photomicrograph, showing granulation tissue and areas of caseous necrosis; × 400.

The thoracic segment of the spine presented an anterior convexity. The twelfth dorsal spinous process was prominent. Tenderness was elicited on palpation of the spine between the eleventh dorsal and the first lumbar vertebra.

The laboratory data were as follows: The Wassermann test was negative; the sputum and the gastric contents did not contain tubercle bacilli. The chemical composition of the blood was normal. Study of the blood revealed nothing significant.

Roentgen examination of the sinuses disclosed a far advanced destructive lesion of the right maxillary antrum. There was evidence of breaking down of the lateral wall and diffuse clouding throughout. There was an abnormal dense protrusion of soft tissue into the oral cavity on this side.

Roentgen study of the spine showed destruction of the lower border of the second lumbar vertebral body and of the upper border of the third. Most of the lateral border of the intervertebral disk was destroyed.

The antrum was irrigated through the inferior meatal opening. Large lumps of foul-smelling white pus were returned with the irrigating fluid. Three months after admission polypoid masses protruded from the artificial opening, and granulations were noted on the nasal floor in this region. Because of the persistent swelling of the soft tissues, the enlarged glands in the neck and the polypi, malignant disease was suspected. The granulations and the polypi were examined histologically. Dr. Oscar Auerbach, the pathologist, reported them as nonspecific granulation tissue and polypi, respectively.

At the end of August, despite frequent irrigations, there was no improvement. The patient now evidenced some weakness of the right external rectus muscle. Roentgen examination at this time showed an extension of the disease into the floor of the orbit, and the right frontal sinus appeared cloudy.

On Sept. 21, 1937, the maxillary cavity was explored by means of an incision in the area of the canine fossa. Necrotic bone was found at the bottom of the

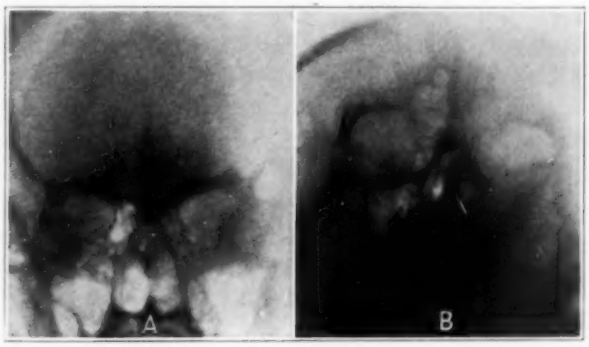

Fig. 2.—*A*, anterior-posterior view of the skull, showing the destruction of the floor and the lateral aspects of the right orbit, as well as the involvement of the right frontal and ethmoid sinus. *B*, roentgenogram made from chin to nose, showing more clearly the destruction of the floor of the orbit.

fistulous tract previously mentioned. The antrum was filled with granulation tissue and polypi. These were removed and sent to the pathologist. Examination of the tissue revealed tuberculosis of the bone and tuberculous granulation tissue.

After operation the patient showed no improvement. The antrum was subjected to ultraviolet irradiation by means of a "cold quartz" applicator through the gingivolabial and the nasal opening. Three times a week aspiration yielded 2 to 3 ounces (59 to 88 cc.) of pus. Occasionally, large sequestrums were removed from the nasal fossa.

On Jan. 10, 1938, the patient complained of diminishing vision of the right eye and of the sudden onset of severe occipital and nuchal pains. Roentgen examination showed marked destruction of the floor of the orbit, with protrusion of the soft parts into the orbit as well as involvement of the right ethmoid and frontal sinuses. Because of the poor general condition of the patient as well as the marked extension of the disease, extensive resection was not thought justified.

REVIEW OF THE LITERATURE

In June 1907 Gleitsmann [1] reviewed the literature on tuberculosis of the antrum. He collected 20 cases. The reports usually indicated that the disease was the result of extension from a neighboring focus. In 12 cases the condition was secondary to tuberculosis of the nasal bones or maxillas, while in the other 8 it had no such origin. The majority of patients had pulmonary tuberculosis. Few of them had symptoms referable to the antrum. In each case tubercle bacilli were found in the material taken from the antrum.

In 3 of the cases no other tuberculous focus was found. These cases were recorded by Coakley,[2] Gaudier [3] and Keckwick.[4] Each author reported a cure after removal of bone and curettage. In 2 other cases reported there was a favorable issue. In the first, reported by Neufeld,[5] the condition originated in the alveolar process and extended into the antrum, forming a fistulous tract. The area was curetted; sequestrums were removed, and the lesion healed. The other was reported by Demme.[6] His patient presented old lupus of the septum, a swelling of the face and a fistula extending from the socket of the first molar tooth to the antrum. This condition cleared after repeated scraping and irrigation.

Gleitsmann concluded from his survey that the prognosis was generally unfavorable, that the treatment was unsatisfactory and that only radical surgical treatment was likely to be of help.

Infection of the maxillary sinus by the tubercle bacillus is a rare condition. I have found 6 cases in the literature since Gleitsmann's report in 1907.

In 1908 Guyot [7] reported the case of a woman who died of miliary tuberculosis seven months after radical antrotomy. Five years later Dorner [8] reported a case in which the condition was thought to be

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secondary to intranasal tuberculoma. Collet's [9] case was observed in 1919 but not reported until 1927. His patient was suffering from pulmonary tuberculosis. The maxillary lesion was secondary to a diseased tooth. After the extraction of the tooth severe lancinating pain developed in the right jaw. Subsequently a fistula from the antrum to the socket of the tooth made its appearance. A Caldwell-Luc operation was performed, but the patient died three weeks later. Jones [10] in 1928 reported the case of a patient with an open pulmonary lesion. He had frequent slight nosebleed. Roentgen examination showed a cloudy antrum with erosion of the superior and inferior borders. Malignant change was suspected; a Caldwell-Luc operation was performed, and a tubercle was observed on the mucosa. Examinations of tissue and pus revealed no specific infection. The diagnosis was made when some acid-fast organisms were found in the antral washings a month after operation. The author claimed cure after the antrum was filled with 50 per cent chaulmoogra oil. Mill's [11] case, observed in 1934, was that of a 23 year old man suffering from laryngeal tuberculosis; the sputum was normal, and a pulmonary roentgenogram showed no abnormality. Because an antral discharge failed to clear up a Caldwell-Luc operation was performed. Examination of the tissue obtained at operation revealed tuberculosis. The last case was reported in 1934 by Burruano.[12] His patient's condition was secondary to an alveolar abscess.

In 1931 Fallas [13] reviewed the literature and discussed the mode of infection. He stated that the maxillary sinus may become infected in the following ways by the tubercle bacillus:

1. By continuity from an infection originating in the eye, the nose or the teeth. Tuberculosis of the eye is rare and was not found in any of the reported cases; it may therefore be discounted as a source of infection. Tuberculosis of the nose is uncommon, but lupus does occur in the nose, and in 2 of the cases reviewed by Gleitsmann infection was secondary to this condition. Extension from a focus in the teeth, particularly from the premolar or molar teeth, is the commonest. Cases of

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this type of infection have been reported by Rethi,[14] Neumayer,[15] Brindle,[16] Weinberger,[17] Koschier [18] and Collet.[9]

2. By hematogenous infection. This presumably is the mode of infection in cases in which there is no neighboring focus of infection. Examples of this are the 3 cases reported by Gleitsmann as primary instances of involvement and the case reported here. Substantiation for the concept of a hematogenous route of infection was furnished in my case by the presence of the spinal lesion.

Fallas described three forms of the disease from the pathologic point of view: (1) the larval, (2) the chronic sinusitic with fistula and (3) the hyperplastic or tuberculomatous.

In the larval form there is only a mucosal infection, and the condition appears as benign sinusitis. The antrum is filled with boggy pale mucosa and polypi. The diagnosis in this type is extremely difficult and can be made only by histologic study of the tissues removed at operation. Riecke [19] emphasized the difficulty in differentiating lesions of this type resulting from foreign bodies in the antrum from true tuberculous lesions.

When the infection results in frank chronic sinusitis with involvement of the bone and formation of fistulas, the diagnosis is easy. Tubercles are readily found. Usually there is a coexistent pulmonary lesion. My case furnishes an exception to this rule.

Tuberculoma of the antrum is extremely rare. Only 1 case of this type has been reported, the case of Guyot.[7] His patient had intranasal tuberculoma. A diagnostic puncture of the antrum encountered what was interpreted as a mass in the antrum. Through a window in the inferior meatus the nasopharyngoscope revealed a tuberculoma. A radical operation produced a good result. The patient died seven months later of a miliary spread of the disease. The tuberculoma is essentially a mucosal lesion and should not be thought of as a distinct type.

SUMMARY

A case of tuberculosis of the antrum resulting from hematogenous infection is reported, because of the unusual nature and rarity of the condition.

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14. Rethi, L.: Ein weiterer Fall von Tuberculose der Kieferhöhle, Wien. med. Presse **40**:2129, 1899.

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16. Brindle, cited by Fallas.[13]

17. Weinberger, M.: Ueber fortgepflanzte Tuberculose der Kieferhöhle, Monatschr. f. Ohrenh. **27**:151, 1903.

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19. Riecke, H. G.: Zur Fremdkörper-Tuberkulose der Kieferhöhle, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:337, 1930.

The diagnosis of tuberculosis of the sinus is difficult when only the mucosa is involved; however, it is easy in the frankly chronic state, in which there is destruction of bone and fistula formation. In this form it must be differentiated from a malignant process because of the regional glandular involvement and the fistula formation.

The prognosis in cases of tuberculous sinusitis is poor. Favorable outcome has been claimed in only 6 of the 26 reported cases.

Successful treatment of this condition depends on early diagnosis and radical surgical intervention. The general condition of the patient also is an influencing factor.

METASTATIC HYPERNEPHROMA OF RIGHT FRONTAL, ETHMOID AND MAXILLARY SINUSES

CHARLES E. CONNOR, M.D., ST. PAUL

Metastatic hypernephroma of the accessory nasal sinuses is, judged by the literature, of infrequent occurrence; this case is reported in the hope that it may, therefore, be of some interest.

REPORT OF A CASE

The patient, a white woman aged 52, first seen on Jan. 25, 1934, complained of a swelling above the right eye and a bloody purulent discharge from the right nostril.

The family and the personal history were completely irrelevant.

The patient had had an infection of the head in November 1933, with profuse bloody purulent discharge, especially from the right nostril. The infection apparently subsided, but a month later a slowly increasing swelling appeared above the right eye; the nasal discharge, both anterior and posterior, persisted, but there was no pain or headache. The patient thought the vision of the right eye was slightly impaired. Except for occasional discomfort in the left upper quadrant of the abdomen and in the back, so slight that no attention had been paid to it, the patient was feeling well.

The physical examination showed no abnormality except the local condition. Swelling and ptosis of the right upper eyelid concealed the upper half of the cornea; the globe was proptosed and pushed downward, and its motion, while fairly free, presented slight but definite limitation in all directions but especially upward. Diplopia was present on extreme rotation to the left; the second image was faint and close to the first. The pupils were round, regular and equal and reacted directly and indirectly to light and in accommodation.

The firm, elastic swelling of the upper eyelid and the supraorbital margin extended up onto the frontal bone for 2 cm., was tender on palpation, fluctuated as though containing fluid under pressure and presented distinct pulsation without bruit; the frontal bone was not tender. The nasal half of the supraorbital margin was destroyed.

The right nostril was filled with bloody purulent crusts; the middle meatus, completely closed by swelling of the lateral nasal wall, bled at the slightest probing, so that exact examination was not possible. The left nostril, the postnasal space, the pharynx and the cervical glands were normal; the right antrum wash was returned clear.

Roentgenograms revealed increased density of all the right sinuses and erosion of the floor of the frontal sinus.

Laboratory examination, including the Wassermann test, showed no abnormality except occasional red and white cells and hyaline casts in the urine.

The tentative diagnosis was chronic suppuration of the right frontal, ethmoid and maxillary sinuses, with erosion of the anterior and posterior plates and the floor of the frontal sinus; the possibility of extradural abscess and abscess of the frontal lobe was considered.

An external operation with the patient under general anesthesia was done on Feb. 2, 1934; the following is the operative record: "The usual incision was made through the eyebrow and about the inner canthus; elevation of the soft tissues revealed a pulsating tumor protruding through an extensive defect in the anterior wall and floor of the frontal sinus. During an attempt to elevate the periosteum from the lacrimal bone and paper plate the curet slipped through the latter structure and entered deeply into the ethmoid sinus, with resulting severe hemorrhage; this was controlled with difficulty. The pulsating mass, found to be the mucous membrane of the frontal sinus, was removed apd a large defect found in the posterior plate of the sinus, through which the dura over the frontal lobe could be seen pulsating. Resection was done in all directions in an effort to reach healthy bone; how successful this was could not be determined, especially in the region of the posterior-inferior angle of the sinus. There was no inter-

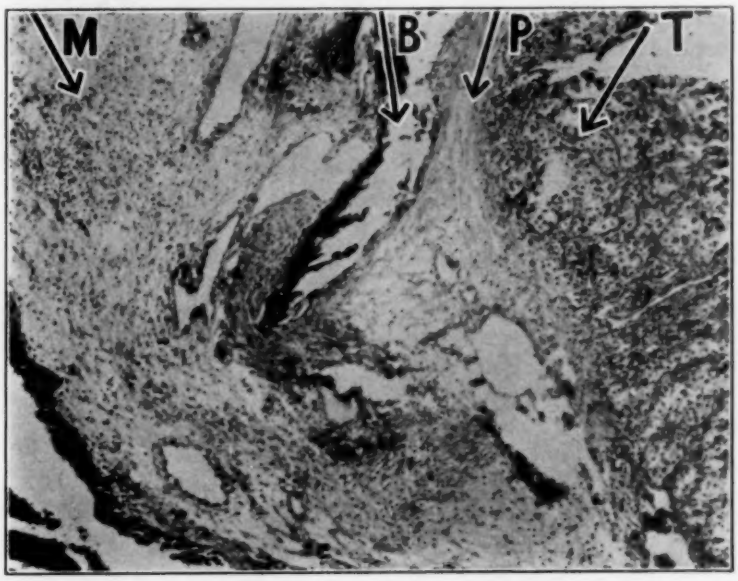

Fig. 1.—Photomicrograph of a metastatic hypernephroma of the frontal sinus, indicative of perforation of the external table of the frontal sinus and involvement of the periosteum. *M* indicates mucosa; *B*, bone; *P*, periosteum, and *T*, the tumor.

sinal septum. The mucosa of the left frontal sinus, apparently thickened and diseased, was removed through the incision over the right sinus; the bone appeared healthy. Recurring hemorrhage prevented further exenteration of the ethmoid sinus. The wound was packed with iodoform gauze and left wide open."

The preoperative diagnosis was reaffirmed without suspicion that the process was malignant. It was noted, however, that the mucous membrane, while much thickened, was friable and somewhat translucent. The following laboratory report was, therefore, a distinct surprise: "Grossly the specimen consists of pieces of tissue removed from the frontal sinus. There are a few small grape-like polypoid masses, but the bulk of the material is fairly solid hemorrhagic, meaty tissue, incorporating white solid tumor-like growth. There is no gross evidence of suppuration. Microscopically the mucosa of the sinus is markedly

edematous and infiltrated by a small number of mononuclear cells and shows areas of hemorrhage. The mucosa rests on a thin bony plate, with fibrosis of the intervening area. A mass of epithelial cells in alveolar nests, showing relatively small, dark-staining nuclei and wide, clear, somewhat foamy cytoplasm, appears to grow out of the inner lining of the bony plate. Numerous areas of hemorrhage within the mass show organization and fibrosis. The diagnosis is hypernephroma of the frontal sinus, probably metastatic."

A pyelogram made during convalescence was reported as follows: "The pyelogram shows the right kidney to be normal; the left is elongated, with deformity of the inferior calix and considerable rounded enlargement of the lower half of the kidney, indicating a tumor in the lower pole. The diagnosis is neoplasm in the lower pole of the left kidney."

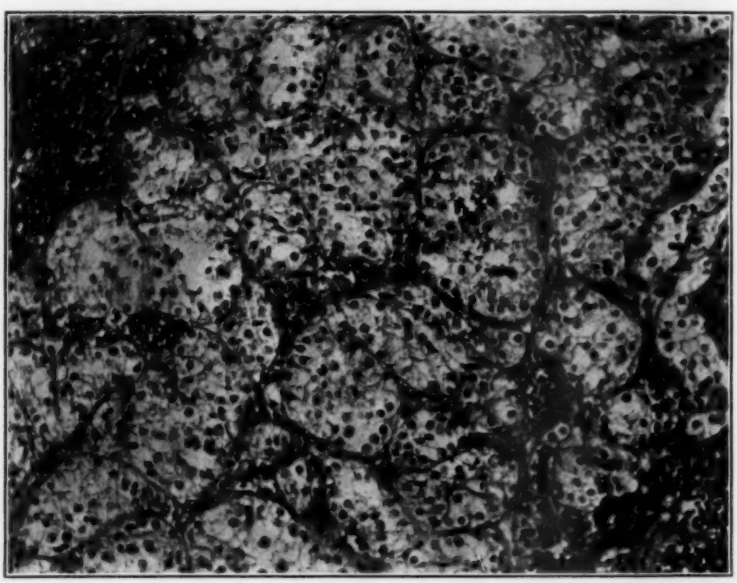

Fig. 2.—Photomicrograph of a metastatic hypernephroma of the frontal sinus, showing characteristic foam cells of the neoplasm.

The obvious diagnosis was now hypernephroma of the right frontal and ethmoid sinuses, metastatic from a hypernephroma of the left kidney; no tumor in the kidney could be palpated, and repeated examination showed the urine to be normal except for occasional red and white cells and hyaline casts.

The patient was transferred to the Cancer Institute of the University of Minnesota for radiation therapy, a summary of which is here given: On February 18, 45 mg. of radium in two capsules, with a filter of 0.5 mm. of silver plus 1 mm. of brass plus 0.5 mm. of rubber, was applied for twenty-four hours (a total of 1,080 mg. hours) inside the right frontal sinus.

The patient received nine roentgen treatments to the region of the left kidney between February 21 and March 14. A total of 1,000 roentgens (as measured in air) was given to a 10 by 20 cm. anterior field, the same amount to a similar posterior field and about 800 to a 12 by 16 cm. lateral field. The factors used were 200 kilovolts and 20 milliamperes, filtered with 1 mm. of copper plus 2.5 mm. of

aluminum, at a target-skin distance of 60 cm. for the lateral field and of 70 cm. for the two other fields.

The frontal sinus received four treatments between March 16 and 24. Two treatments were given to an anterior field and two to a right lateral field; each field measured 7 by 10 cm., and each received a total of 880 r. The factors were those noted previously, except that a filter of 0.5 mm. of copper plus 2.5 mm. of aluminum was used and the target-skin distance was 60 cm.

The local process appeared to subside, but the incision remained open at the nasal end. The cavity of the frontal sinus appeared to granulate slowly; the intranasal condition remained unchanged.

Severe nasal hemorrhage occurred on August 21, September 1 and September 4. Roentgen studies on September 13 revealed "considerable destruction of a rather peculiar character in the region of the right maxillary sinus; the appearance suggests some type of destructive process, probably a tumor. There is no evidence of hypernephroma."

Nine months after the operation, recurrence of the hypernephroma in the frontal sinus was evident; the wound showed no evidence of healing, and a small bud of what appeared to be granulation tissue appeared in the nasofrontal duct. The right nasal cavity was completely plugged with a firm grayish red mass, suggesting involvement of the ethmoid sinus and the antrum in the malignant process and substantiating the roentgen report of September 13; repeated biopsy of this tissue showed only granulation tissue.

A severe nasal hemorrhage on November 2 necessitated hospitalization; on November 3 the right external carotid artery was ligated, and on November 9 a radical operation was performed on the antrum. The membrane was much thickened, and a moderate amount of pus was present; biopsy showed hypernephroma.

Severe nasal hemorrhage occurred on December 13. On Jan. 11, 1935, four gold implants, with a total of 4.8 millicuries, were inserted in the area of the right frontal sinus. The thickness of the wall was 0.3 mm. of gold and the total dose 630 millicurie hours.

Late in January pain developed in the right shoulder and left leg; roentgenograms showed no lesions, and neurologic examination revealed no abnormality.

The pain persisted and increased in severity, involving the left leg, thigh and hip. Tenderness developed over the left great trochanter; a roentgenogram showed an area of diminished density in the left pubic bone.

In late February evidence of irritation and beginning involvement of the sacral plexus appeared (positive Laségue sign), and in April a tumor appeared on the left thigh and increased slowly in size. A roentgenogram showed an area of destruction in the distal third of the left femur, evidently due to metatasis, with fracture and callus formation; the other bones were normal. Physical examination showed a pathologic fracture in the lower portion of the left femur, with marked lateral angulation of the lower fragment on the upper; crepitation was present.

The patient gradually lost weight and strength, became septic and died on May 8.

Autopsy showed recurrence of the tumor in the right frontal sinus (the ethmoid sinus and the antrum were not examined) and generalized abdominal metastasis, involving practically all the viscera; pathologic fracture of the left femur, due to metastasis, was present.

An extended discussion of hypernephroma is not within the scope of this report, but it may be mentioned in passing that agreement as to the origin of this tumor has not been reached; opinion as to the tissue of

origin is divided between adrenal rests (Grawitz) and renal epithelium in various stages of development. All writers agree on the tendency of hypernephroma to metastasize, especially to the lungs, liver, pelvis and skeleton, particularly the skull. MacCallum [1] cited a case in which metastasis occurred in the diploe of the bones of the skull.

However, metastasis to the sinuses is not common, if one is to judge by the literature; only 2 cases have been reported in the last ten years. Von Eicken [2] reported a case in 1934 which possessed several points in common with that reported here. The tumor appeared suddenly, pulsation and crepitation were present and a roentgenogram showed erosion of the floor of the frontal sinus. Operation was rendered slow and difficult by profuse bleeding, which required the ligation of many vessels.

Von Eicken's patient was given two high voltage roentgen treatments and 1,000 milligram hours of radium irradiation, needles being inserted directly into the cavity of the tumor; the response was apparently satisfactory, although the final outcome was not given. The author stated that metastatic hypernephroma of the sinuses is uncommon and that it usually responds to irradiation; the primary hypernephroma is not radiosensitive.

Menger and Arons [3] reported a case of metastatic hypernephroma of the tonsil in which removal with tenaculum and snare was followed by radiation therapy of the tonsil after the technic of Coutard; they commented on the friability of the tumor tissue.

Carnevale-Ricci [4] reported a metastatic hypernephroma of the frontal sinus, without symptoms, which was discovered only at operation; the operation was bloody. The primary renal lesion was not diagnosed.

Study of this case yielded no information as to the exact point of the initial lesion; the extensive involvement of both the anterior and the posterior wall and the floor of the frontal sinus would point to this cavity rather than to the ethmoid sinus as the site of the initial metastasis, although the latter was undoubtedly involved when the patient was first seen. This extensive involvement of bone, together with the recognized tendency of hypernephroma to involve the bones of the skull, suggests the probability that the frontal bone itself, rather than its lining mucosa, was first involved.

Dr. Horace Newhart, Professor of Otolaryngology in the University of Minnesota Medical School, gave me access to the records of the University Hospital, and Dr. T. E. Broadie, Superintendent of Ancker Hospital, St. Paul, permitted me to study the postmortem records.

25 West Fourth Street.

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Clinical Notes

USE OF OSCILLOGRAMS IN STUDIES OF THE VOICE

ROBERT B. TAFT, M.D., CHARLESTON, S. C.

The value of the oscillographic tracing in the study of vocal characteristics is well known, but the acquisition and management of the equipment appear to create a rather formidable problem for one who has little familiarity with such apparatus.

Today the microphone and the public address system are so commonly seen that they are seldom noticed. The cathode ray oscillograph is now in common

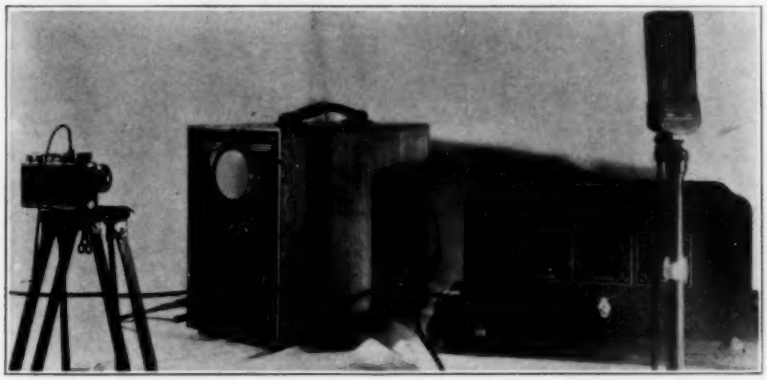

Fig. 1.—Apparatus used in the work. Right to left: microphone, which picks up the voice; public address system, which builds up the power to the necessary intensity; oscillograph, on whose screen the patterns appear; camera, for securing a permanent record of the patterns.

use in practically every well equipped radio repair shop. The combination of these instruments with a good miniature camera is all that is necessary for the making of excellent tracings.

The purpose of this brief paper is to point out that any physician can, with the cooperation of a skilled radio repair man, experiment in this work at nominal cost.

Figure 1 shows the arrangement of the apparatus; figure 2 shows tracings of the soprano voice with two different degrees of laryngeal pressure.

The following is the method of procedure: Any camera may be used which is capable of close-up focusing without supplementary lenses; the greater the lens aperture available, the faster the exposure may be made. Fast exposure is a great advantage, as many of the patterns are rather unsteady. With a fairly

fast film, a lens aperture of f 2 permits exposures of 1/200 to 1/500. These factors give a negative with dark shadows and white high lights, which may be enlarged to any reasonable size.

As most of the public address systems give much greater amplification than needed for this work, as small a one as possible should be chosen and the power output turned to the lowest usable point. It is desirable that the microphone be

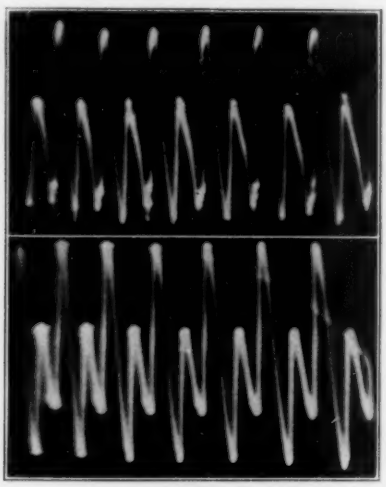

Fig. 2.—Above, typical pattern of the soprano voice. Below, the same, with slightly different laryngeal pressure.

placed rather close to, and to one side of, the singer's lips, so that the breath does not strike it.

In all cases the pattern may be studied at leisure on the screen before any photographs are made.

105 Rutledge Avenue.

AN EFFICIENT AND PRACTICAL BOOTH FOR TESTING HEARING

HORACE NEWHART, M.D., AND HENRY E. HARTIG, PH.D., MINNEAPOLIS

The need for excluding distracting and masking noises to insure accuracy in making hearing tests has been appreciated and emphasized only in recent years. In the presence of noises which ordinarily prevail in downtown office buildings or schools it is impossible to detect or measure slight but significant degrees of loss of hearing. Their early detection according to modern otologic practice is of great importance, especially in the field of the prevention and amelioration of deafness.

Presented before the Section on Laryngology, Otology and Rhinology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 17, 1938.

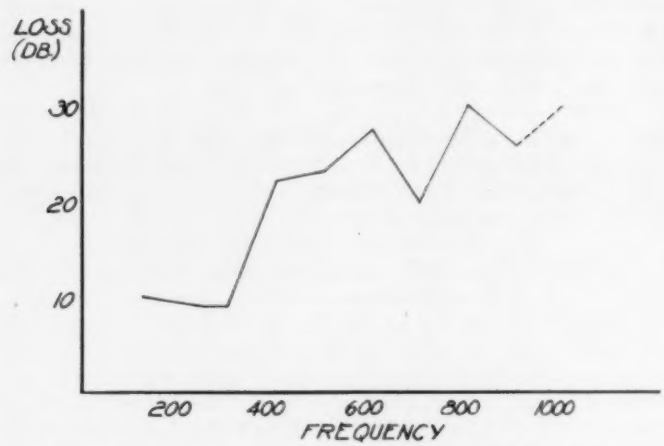

Fig. 1.—Acoustic loss in transmission through the audiometric booth.

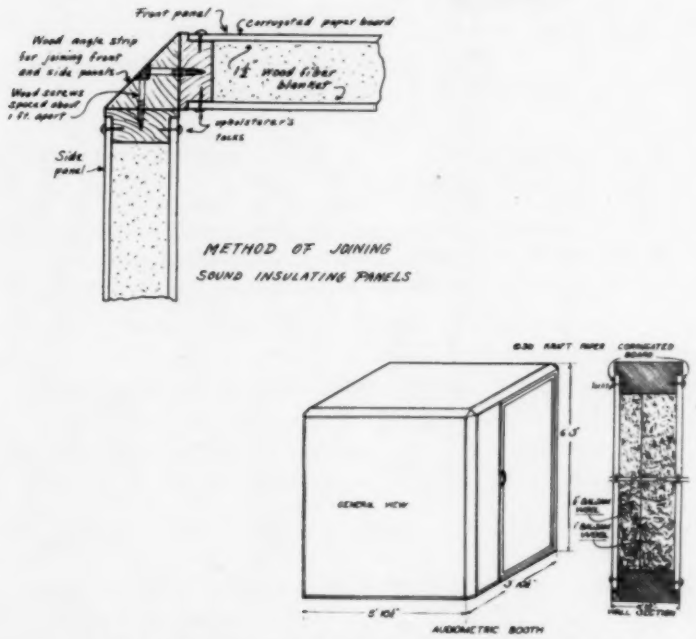

Fig. 2.—Construction and method of assembly of the audiometric booth.

It is impossible to construct rooms absolutely soundproof against disturbing noises of unlimited intensity, and even to obtain total isolation from noises of moderate intensity requires massive construction and a large expenditure. For accurate clinical work it is necessary, nevertheless, to reduce the prevailing noise to a practical minimum. This as a rule is not accomplished by the carpenter or contractor not experienced in acoustic construction.

To meet the necessity for a hearing chamber providing substantial isolation, we initiated a program of investigation extending over a period of two years and conducted as a joint project of the departments of otolaryngology and electrical engineering at the University of Minnesota. The object in view was to construct a practical, efficient, portable and inexpensive booth, suitable for use in physicians' offices, schools and other places where hearing tests are made.

Trial test panels were built, embodying various materials and forms of construction, and acoustic transmission tests were conducted thereon. The form finally adopted embodies to a large extent the desirable features sought. It consists of outer reflecting layers of double-faced corrugated Kraft paper board, between which is held a 1½ inch (4 cm.) double layer of wood fiber blanket, the whole held in wooden frames, which compose the side and roof panels of the booth. The structure rests on a base of 1 inch (2.5 cm.) absorbent wood fiber board.

The desirable features obtained by this type of construction include:

1. A relatively high effectiveness in reducing noise.
2. Extreme lightness. (The entire structure weighs only 250 pounds [113 Kg.]—shipping weight 400 pounds [181 Kg.].)
3. Knock-down construction.
4. Transportability.
5. Simplicity and ease of erection. (Two men with no other tools than screw drivers can readily erect the booth in thirty minutes.)
6. Low cost.

In figure 1 is shown the reduction in loudness obtained when pure tones of various frequencies are transmitted from the outside to the inside of the chamber. Complex sounds, such as the noises produced by vacuum cleaners, buzzers or speech, suffer a reduction on the average of 23 decibels. In general it may be said that the chamber is most efficient in attenuating high-pitched sounds.

The form of construction and the method of assembly are shown in figure 2.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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ADVANCES IN THE FIELD OF ALLERGY AS RELATED TO OTOLARYNGOLOGY DURING THE YEAR 1938

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AND
C. M. KOHN, M.D.
KANSAS CITY, MO.

This paper is the fourteenth review of the available literature on allergy as related to otolaryngology made by one of us in the ARCHIVES. The reader may have noticed the rapid advance in knowledge of the subject during this period. Especially interesting is the fact that a gross majority of original contributions have been made from the private offices of physicians who practice in this field.

CAUSES OF ALLERGY

Foods.—Wightman [1] reports a case of asthma due to sensitivity to soybean which followed inhalation or ingestion of this agent. One of us [2] reported 5 cases of sensitivity to soybean dust in 1934. This subject is of particular interest because of the widespread use of soybean.

Loveman [3] definitely established sensitivity of the skin and mucous membrane to oil of anise. In addition to eliciting a positive reaction to a patch test with oil of anise, he was able to reproduce stomatitis by having the subject chew candy flavored with oil of anise. There was no reaction after the swallowing of capsules containing oil of anise.

Molds.—Rackemann, Randolph and Guba [4] report 4 cases of sensitivity to a specific mold, Cladosporium fulvum Cke. In each case asthma

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1. Wightman, H. B.: Soybean Sensitivity, with a Case Report, J. Allergy **9:** 601, 1938.

2. Duke, W. W.: Soybean as a Possible Important Source of Allergy, J. Allergy **5:**300, 1934.

3. Loveman, A. B.: Stomatitis Venenata: Report of a Case of Sensitivity of the Mucous Membranes and the Skin to Oil of Anise, Arch. Dermat. & Syph. **37:** 70 (Jan.) 1938.

4. Rackemann, F. M.; Randolph, T. G., and Guba, E. F.: The Specificity of Fungus Allergy, J. Allergy **9:**447, 1938.

followed exposure to the spores of this mold, growing in a commercial tomato greenhouse. Intradermal tests produced strong positive reactions to C. fulvum Cke. but little or no reaction to other species of Cladosporium. This would indicate a high degree of specificity among the species of the genus Cladosporium.

Simon [5] reports a case of conjunctivitis in which seasonal variation in the patient's symptoms coincided with seasonal variation in the spore content of the air. Change in geographic location gave relief. The reaction to cutaneous tests with fungus extract was positive. The application of fungus extract to the conjunctiva during a temporary recovery reproduced the illness. Desensitization with fungus extract gave relief.

Chemicals.—Goodman and Sulzberger [6] report 12 cases of irritation of the lips and 2 of dermatitis of the face caused by lipstick. In each case they demonstrated sensitivity to coloring material contained in the lipstick. The indelible dyes of the bromfluorescein group were the most frequent offenders and produced positive reactions in patch tests. In a number of tests positive reactions were elicited also by nonindelible lake colors. The fatty ingredients of the colorless lipstick base and the perfumes were eliminated as etiologic factors. The authors think that sensitivity to dye is definitely based on an allergic mechanism.

Sézary and Horowitz [7] collected 11 cases of cheilitis caused by lipstick. Eosin proved to be the etiologic factor.

Hallier [8] describes a case of dermatitis which followed the application of lipstick. The patient reacted to many different brands of lipstick but when tested with their various ingredients was found to be sensitive to eosin alone.

Biederman [9] reports 19 cases of allergy caused by the use of Drene shampoo. The symptoms were sneezing, rhinorrhea, asthma, eczema and dermatitis. In each instance the illness disappeared after withdrawal of the offending shampoo.

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5. Simon, F. A.: Allergic Conjunctivitis Due to Fungi, J. A. M. A. **110**:440 (Feb. 5) 1938.

6. Goodman, J., and Sulzberger, M. B.: Acquired Specific Hypersensitivity to Simple Chemicals: I. Eczematous Sensitivity to Clothing and to Cosmetics, with Special Reference to Dyes, J. Allergy **9**:136, 1938.

7. Sézary, A., and Horowitz, A.: Le cheilite du rouge, Presse méd. **45**:137, 1937.

8. Hallier, F. F.: Lipstick Dermatitis, with a Report of a Case Due to Eosin, Brit. J. Dermat. **49**:485, 1937.

9. Biederman, J. B.: Sensitivity to Drene Shampoo, New England J. Med. **217**:1088, 1937.

O'Leary and his associates [10] report a case of urticaria and eczema following the injection of iodobismitol with saligenin. They were able to demonstrate by patch testing that saligenin, a local anesthetic, caused the eruption.

Drugs.—Cohen and Simon [11] report 2 cases of "insulin hypersensitivity." In 1 of these the patient was hypersensitive to proteins of the animal from which the insulin was obtained; in the other, to an extraneous substance contained in commercial insulin. This type of sensitivity is not rare. Many cases have been reported in the literature during the past few years. The exact cause of the phenomenon was rarely proved. It would seem to us that hypersensitivity to the active principle of insulin ought to be incompatible with life and that the allergic reaction that follows its use must be due always to some other substance in the preparation.

Finney [12] reports a case of severe dermatitis medicamentosa, and Myers, Vonder Heide and Balcerski [13] one of severe exfoliative dermatitis, following the administration of sulfanilamide. Recovery followed withdrawal of the drug in each case.

The increasing popularity of sulfanilamide therapy has emphasized the fact that it is highly toxic to a small minority of patients. This drug should be suspected of causing any unusual illness that follows its administration.

Miscellaneous Agents.—Jameson [14] reports a case of nasal allergy caused by the housefly (Musca domestica). The patient reacted positively to scratch, intracutaneous and passive transfer tests. One of us has studied a case in which local, constitutional and febrile reaction followed the bite of houseflies.

Kern [15] reports a case of asthma due to sensitization to a species of mushroom fly (Aphiochaeta agarici). An extract of the flies not only produced positive reaction in cutaneous tests but was efficacious in treatment. Specific reagin in the patient's serum was demonstrable by passive transfer tests.

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10. O'Leary, P. A.; Montgomery, H.; Brunsting, L. A., and Bailey, R. J.: Sensitivity to Saligenin (Salicyl Alcohol), a Local Anesthetic Used in Iodobismitol (Squibb), Arch. Dermat. & Syph. **35**:982 (May) 1937.

11. Cohen, A. E., and Simon, F.: Insulin Hypersensitivity, J. Allergy **9**:503, 1938.

12. Finney, J. O.: Severe Dermatitis Medicamentosa Following the Administration of Sulfanilamide, J. A. M. A. **109**:1982 (Dec. 11) 1937.

13. Myers, G. B.; Vonder Heide, E. C., and Balcerski, M.: Exfoliative Dermatitis Following Sulfanilamide, J. A. M. A. **109**:1983 (Dec. 11) 1937.

14. Jameson, H. C.: The House Fly as a Cause of Nasal Allergy, J. Allergy **9**:273, 1938.

15. Kern, R. A.: Asthma Due to Sensitization to a Mushroom Fly (Aphiochaeta Agarici), J. Allergy **9**:604, 1938.

Stevens and Jordani [16] report 5 cases of sensitization to jute, a vegetable fiber grown in India and used for making burlap, gunny sacks, cheap furniture, rugs and carpets. Because of this widespread use, a wider incidence of this sensitization would be expected.

Derris root, a tropical shrub used as an active ingredient in insect powder, is described by Weston [17] as the cause of bronchial asthma in 2 cases.

DIAGNOSTIC METHODS

Sherman, Kaplan and Walzer [18] induced sensitization in the nasal mucous membrane of the inferior turbinate and septum of normal persons by injecting a reagin-bearing serum obtained from patients with ragweed pollenosis. The left side of the nose was regularly chosen for sensitization, and the untreated right side served as a control. The investigators were able to elicit a reaction at the sensitized sites by intravenous, subcutaneous and intramucosal injections and by topical application of the antigen, in that order of effectiveness.

In 1932, Todd and his associates [19] called attention to the significance of the roentgen finding of transverse lines of increased density in the epiphysial ends of the long bones in children. These scorings disappear within a couple of years. They are most frequently seen in the lower end of the tibia and radius and are most common in children up to 6 years. Todd expressed the opinion that these scorings develop as a result of temporary abolishment of growth because of a metabolic disturbance. The mineral arriving at the growing end in the usual amount is laid down in a smaller area, which contains more mineral than the adjacent bone and thus casts a denser shadow on the roentgenogram. Such scorings differ from those produced by infectious disease and injury in that they do not run across the entire width of the bone and are therefore narrower and less dense. Cohen and Friedmar [20] analyzed 805 examinations of children, ranging from 2½ years to 16. Of the 610 who had definite clinical signs of allergy, 361 (59.2 per cent) presented scorings and 249 (40.8 per cent) did not. Of the 195 who lacked clinical signs of allergy, 72 (36.9 per cent) presented scorings of 123 (63.1 per cent) did not.

16. Stevens, F. A., and Jordani, L.: Sensitization to Jute, J. Allergy **9**:610, 1938.

17. Weston, C. G.: Derris Root—A New Allergen, J. Allergy **9**:62, 1937.

18. Sherman, H.; Kaplan, C., and Walzer, M.: Studies in Mucous Membrane Hypersensitiveness: II. Passive Local Sensitization of the Nasal Mucous Membrane, J. Allergy **9**:1, 1937.

19. Growth and Development of the Skeleton, in Growth and Development of the Child: II. Anatomy and Physiology, White House Conference on Child Health and Protection, New York, Century Company, 1932, p. 89.

20. Cohen, M. B., and Friedmar, S.: Scorings in the Long Bones as a Guide to the Management of Food Allergy in Children, J. Allergy **9**:54, 1937.

The authors feel that this is definite evidence of a relation between the scorings and the presence of allergy. They state also that observations over two years enabled them to confirm the presence of scorings in practically every child under 6 years who had demonstrable food allergy. They feel that a child is not well as long as new scorings appear and think that their presence indicates activity of the disease.

Chobot and Merrill [21] in a study made on children found scorings of the bone in a high percentage of both allergic and nonallergic patients. They think that this finding is not constant in those suffering from allergic disease and that it is hazardous to diagnose an allergic state on the basis of it.

We do not feel that this work is out of harmony with that of the former observers. It is not claimed by Todd or Cohen that allergy is the only disease capable of scoring bones in the way described. Scorings mean a retardation of growth, and it is certain that allergy can retard growth to the point of causing dwarfism, gross kyphosis, which can rival that caused by Pott's disease, and distortion of bones of the chest, sternum and face in extreme degree.

In 1934 Vaughan [22] revived Widal's "digestive hemoclasia" test and renamed it the "leukopenic index." He expressed the opinion in substance that it might be a useful addition to the technical study of allergy and stated after exhaustive study that 81 to 93 per cent of patients have leukopenia with a deficiency of 1,000 or more cells after eating a food to which they are hypersensitive. Other foods are inclined to increase the count.

Rinkel [23] reported that he was able to find clinical evidence of food allergy in 90 per cent of the patients with leukopenic response, whereas compatible foods uniformly lead to leukocytosis. There have been many supporters of these conclusions.

In attempting to verify them Loveless, Dorfman and Downing [24] made exhaustive studies of the leukocyte counts of allergic and nonallergic patients before and after the ingestion of foods. Their technic was regulated so as to correct every source of error and allow greatest accuracy in counting. They conclude from their evidence that postprandial leukopenia is not an index of incompatibility to foods. In

21. Chobot, R., and Merrill, E. F.: Bone Scorings in Normal and Allergic Children, J. Allergy **8**:588, 1937.

22. Vaughan, W.: Food Allergens: The Leucopenic Index; Preliminary Report, J. Allergy **5**:601, 1934; Further Studies on the Leucopenic Index in Food Allergy, ibid. **6**:78, 1934.

23. Rinkel, H. J.: The Leucopenic Index in Allergic Diseases, J. Allergy **7**: 356, 1936.

24. Loveless, M.; Dorfman, R., and Downing, L.: A Statistical Evaluation of the Leucopenic Index in Allergy, J. Allergy **9**:321, 1938.

addition, they fail to support the contention that leukocytosis is the normal response to ingestion.

Brown and Wadsworth [25] confirm the results of Loveless, Dorfman and Downing in an accurate clinical and mathematical study of postprandial changes in the blood. They found no adequate proof of the existence of either normal postprandial leukocytosis or anaphylactic postprandial leukopenia. In their series of over 2,000 leukocyte counts they found no physiologic, pathologic or mathematical justification for the use of the leukopenic index.

We are unable to do more than report these results, for we have had little experience with the leukopenic index. Certain it is that conclusions drawn from history, physical examination and clinical cutaneous and passive transfer tests should not be altered because of leukocyte counts in which the changes are so small as to fall within the limit of error due to the fallibility of technicians.

PHYSICAL ALLERGY

Goodson [26] briefly reviews the literature on physical allergy. Of particular interest is the fact that in 50 per cent of the cases reviewed a family history of allergic disorders was elicited. In approximately 25 per cent of the cases the onset seemed to follow an acute febrile illness, and in the majority of cases symptoms appeared in the second or third decade. He discusses the relation of histamine to the mechanism of the reaction and states that the proof of its role in physical allergy is not yet at hand. This paper well presents the present status of the subject.

PATHOLOGY

Cazort and Cook,[27] Rollin [28] and O'Leary [29] report cases of atopic dermatitis associated with opacity in the lenses and present additional evidence leading toward the conclusion that atopic dermatitis with associated juvenile cataract is not coincidental but is an allergic syndrome.

Rosenberg and Rosenberg [30] reported a case of subcutaneous emphysema appearing after an attack of bronchial asthma. They review the

25. Brown, E. A., and Wadsworth, G. P.: The Leucopenic Index, J. Allergy **9**:345, 1938.

26. Goodson, W. H.: Physical Allergy, Proc. Staff Meet., Mayo Clin. **13**: 500, 1938.

27. Cazort, A. G., and Cook, R. C.: Atopic Dermatitis with Rapidly Maturing Cataracts, J. Allergy **9**:521, 1938.

28. Rollin, J. L.: Katarakt bei Neurodermie, Ztschr. f. Augenh. **92**:16, 1937.

29. O'Leary, P. A.; Montgomery, H.; Brunsting, L. A., and Bailey, R. J.: Atopic Eczema with Cataracts, Arch. Dermat. & Syph. **35**:981 (May) 1937.

30. Rosenberg, L., and Rosenberg, J.: Subcutaneous Emphysema Complicating Bronchial Asthma, Am. J. M. Sc. **195**:682, 1938.

literature and analyze 17 previously reported cases. Of interest is the fact that each patient recovered. No treatment except palliative measures for asthma was used.

Thieme and Sheldon [31] from a study of autopsy material state that the following criteria are necessary for a pathologic diagnosis of bronchial asthma:

> (A) Hyaline thickening of the basement membrane of medium sized bronchi; (B) hypertrophy of the musculature of medium sized bronchi; (C) mucinous plugs in large and small bronchi; (D) eosinophilic infiltration; (E) sacculation of the bronchi; and (F) excessive production of mucin and widening of the bronchial glands.

They state that morphine and atropine should never be used for status asthmaticus.

TREATMENT

Kern, Crump, Roddy and Borow [32] have continued their experimental study of the intracutaneous method of administering toxin. They demonstrated a negative reaction to the Dick test after the intracutaneous injection of scarlatinal steptococcus toxin in about one-tenth the dose which would have been required if it had been given subcutaneously. By this dosage they completely avoided the constitutional reaction which rather frequently follows the larger dose administered subcutaneously. The immunity obtained by the smaller dose was not so lasting.

Tuft [33] found that the addition of epinephrine to a mixture containing an antigen or antibody delays absorption of the agent but does not interfere with its specific immunologic effect. The value of this procedure lies in the preventing or minimizing of constitutional reaction.

Naterman [34] describes the use of a pollen extract emulsified in wool fat and olive oil. He states that in this form pollen extract is absorbed less rapidly and larger doses can be tolerated by the patients. Moreover, the number of injections required is reduced about 50 per cent. General reactions are decreased in number by the slow absorption. Clinical results obtained with the extract in emulsion are as good as or better

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31. Thieme, E. T., and Sheldon, J. M.: A Correlation of the Clinical and Pathologic Findings in Bronchial Asthma, J. Allergy **9**:246, 1938.

32. Kern, R. A.; Crump, J.; Roddy, R. L., and Borow, S.: Scarlet Fever Immunization by Intracutaneous Injection of Scarlatinal Streptococcus Toxin, J. Allergy **9**:125, 1938.

33. Tuft, L.: The Influence of Epinephrine upon Antigen and Antibody Action, J. Allergy **9**:454, 1938.

34. Naterman, H. L.: The Treatment of Hay Fever by the Injection of Pollen Extract Emulsified in Lanolin and Olive Oil: Preliminary Report, New England J. Med. **218**:797, 1938.

than those obtained with an extract in solution. Sensitivity as indicated by cutaneous tests was markedly reduced. This study should be continued.

Caulfeild [35] reports the results of treatment of poison ivy dermatitis by intramuscular administration of an extract of poison ivy plant which he terms "Rholigen." He proved the diagnosis in each case by a patch test with "Rholigen" and reduced the positiveness of the reaction to a quantitative patch test by treatment. This reduction coincided with clinical immunity to ordinary contact with the weed.

Swineford [36] treated asthma by prescribing the intrapulmonary inhalation of benzedrine. He placed gauze saturated with 0.5 cc. of pure benzedrine base in a special 6 inch (15 cc.) glass tube, which was then inserted in the mouth almost to the uvula. With the lips closed tightly around it and the nose closed, the patient inhaled deeply. It was demonstrated that inhalation of volatile benzedrine base increases the vital capacity of the lung. Blanching of both the normal and the inflamed bronchial mucosa was noticed. The blanching and shrinking of the latter aids in bronchoscopic inspection. Swineford compares the therapeutic effect of benzedrine in relieving the paroxysms of asthma to that of 1 to 100 epinephrine vapor.

Frankly, we believe that this method of therapy, if it must be used, is more conservative than inhalations of strong solutions of epinephrine. We look on each, however, as strong treatment which might often have a disagreeable aftermath. Each method appears to us a last resort.

Boyer [37] reports on the use of propadrine-hydrochloride in the treatment of allergy. This product is a synthetic substance related to epinephrine and ephedrine. He found that propadrine hydrochloride gives clinical relief comparable to or better than that given by ephedrine, without causing palpitation, tachycardia, nervousness and insomnia.

Criep and Hampsey [38] review literature dealing with the value of iodized oil in the treatment of bronchial asthma. They sent a questionnaire to physicians interested in this type of treatment. Over two thirds of those who replied had abandoned it and showed a lack of enthusiasm for, if not direct opposition to, its use. They feel that only a small, select group of patients, with intractable asthma, are benefited by iodized oil.

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35. Caulfeild, A. H. W.: The Specific Diagnosis and Treatment of Poison Ivy (Rhus Toxicodendron) Dermatitis, J. Allergy **9**:535, 1938.

36. Swineford, O., Jr.: Intrapulmonary Inhalation of Benzidrine, J. Allergy **9**: 572, 1938.

37. Boyer, W. E.: The Clinical Use of Phenyl-Propanol-Amine Hydrochloride (Propradrine) in the Treatment of Allergic Conditions, J. Allergy **9**:509, 1938.

38. Criep, L. H., and Hampsey, J. W.: Therapeutic Value of Iodized Oil in Bronchial Asthma, J. Allergy **9**:23, 1937.

Flamm [39] reports 2 cases of patients treated for asthma by the instillation of iodized poppyseed oil in which the oil was retained for many months in the alveoli of the lungs. A continued attack of asthma was attributed to the retained oil with secondary pneumonitis. Flamm disapproves this form of therapy.

Hunt [40] treated bronchial asthma in 25 cases with comparatively large doses of ascorbic acid (synthetic vitamin C), administered both by mouth and intravenously. He tried this method because ascorbic acid is normally present in the adrenal glands and asthma may follow adrenal hypofunction. He concludes, however, that ascorbic acid is of no value in the treatment of bronchial asthma.

Prickman and Koelsche [41] administered an extract of adrenal cortex intravenously to 19 patients, of whom 16 had asthma, 2 severe vasomotor rhinitis and 1 severe chronic urticaria and angioneurotic edema. Sodium chloride, also, was given by mouth in each case. From the result obtained the authors conclude that this method of treatment is not beneficial. This report harmonizes with several that have appeared in the literature during the past few years.

Hermann and Aynsworth [42] report on the use of theophylline with ethylenediamine in 16 cases of severe asthma in which the usual palliative measures had failed. All were classified as cases of "status asthmaticus." The drug was administered intravenously in doses of 0.24 to 0.48 Gm. diluted to 10 cc. with physiologic solution of sodium chloride. The authors state that of 41 injections given 31 gave prompt and complete relief and that there were only 2 failures. Response to epinephrine was restored in the cases in which it had been lost.

Stoesser and Cook [43] report on the result of treatment of intractable asthma in children with hypertonic dextrose solution administered intravenously. They used 150 to 200 cc. of a 25 per cent solution with an excellent clinical result, which they believe was due to a specific action of dextrose rather than to the effect of a hypertonic solution.

Keeney [44] reports on the treatment of status asthmaticus with a mixture of 100 cc. of 50 per cent sucrose (cane or beet sugar) and 0.5 cc. of

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39. Flamm, G.: Retention of Lipiodol in the Lungs, J. Allergy **9**:593, 1938.

40. Hunt, H. B.: Ascorbic Acid in Bronchial Asthma, Brit. M. J. **1**:726, 1938.

41. Prickman, L. E., and Koelsche, G. A.: Observations in the Treatment of Asthma and Related Conditions with Suprarenal Cortical Extract (Cortin), J. Allergy **9**:158, 1938.

42. Hermann, G., and Aynsworth, M. B.: Successful Treatment of Persistent Extreme Dyspnea, "Status Asthmaticus," J. Lab. & Clin. Med. **23**:135, 1937.

43. Stoesser, A. V., and Cook, M. M.: Hypertonic Dextrose Solutions in the Treatment of Bronchial Asthma, Journal-Lancet **58**:12, 1938.

44. Keeney, E. L.: The Effectiveness of Intravenous Hypertonic Sucrose and Adrenalin in the Treatment of Status Asthmaticus, J. Allergy **9**:497, 1938.

1 to 1,000 solution of epinephrine hydrochloride administered intravenously. He produced immediate relief from asthma which persisted two and one-half to three hours. The number of patients treated is small. We wish to warn against unnecessary intravenous use of epinephrine. It is effective for the moment but may have an aftermath which simulates the reverse of an epinephrine effect. We do not wish to imply, however, that this form of therapy is not indicated for extreme conditions.

Spain and Fuchs [45] state that the perennial method gives the best result in the treatment of hay fever. They recommend the continuance of large doses.

45. Spain, W. C., and Fuchs, A. M.: A Clinical Study of the Advantage of the Perennial Treatment of Hay Fever, South. M. J. **30**:1199, 1937.

News and Comment

CASSELBERRY PRIZE FUND

The sum of $500 has accrued from the Casselberry Fund, for encouraging advancement in the art and science of laryngology and rhinology, and is now available, in part or as a whole, for a prize award or decoration or to defray the expense of original investigation or research in the domains mentioned. Theses or reports of work done must be in the hands of the secretary, Dr. James A. Babbitt, 1912 Spruce Street, Philadelphia, before February 1 of any given year. Any further information desired will be gladly furnished by the secretary.

Abstracts from Current Literature

Ear

THE DIAGNOSIS OF DISEASES OF THE NEURAL MECHANISM OF HEARING BY THE AID OF SOUNDS WELL ABOVE THRESHOLD. E. P. FOWLER, Laryngoscope **47**:289 (May) 1937.

Fowler shows that the only way to determine in the living the presence, extent and type of auditory neural lesions is to test the hearing for sounds of various frequencies and intensities. There are many means for doing this, but every method yet devised is frequently uncertain and often misleading, even though its limitations are appreciated. The author mentions the tests most commonly relied on for diagnosis and points out their shortcomings. He then calls attention to the fact that any or all of the standard tests may appear positive and yet there may be no nerve deafness, that all are fundamentally dependent on bone conduction for diagnostic significance and that bone conduction is often unreliable. The method the author described last year makes it impossible to differentiate neural from conduction deafness without recourse to either air conduction or bone conduction threshold measurements. It is the only reliable test for neural deafness which does not require confirmation by other tests. The test consists in measuring the recruitment of loudness with intensity—by balancing the loudness of sounds—thrown alternately first into one ear and then into the other (the alternate binaural loudness balance). The varying differences in loudness necessary for balance at the various steps above threshold are the measures of the rates or recruitment of the neural elements necessary for equal balance. They are therefore a measure of the location, number and extent of loss of the neural elements which initiate, conduct or relay impulses from the end organs to the cortex. The author briefly restates the fundamental observations described in detail last year. The phenomena described occur in varying degrees at different frequencies and vary with certain types of curves. The author observed several apparent exceptions, which he describes. In conclusion, he shows a series of graphs, which clearly demonstrate how at any frequency the alternate binaural loudness balance measures the recruitment of loudness in deafness from various combinations of lesions of the middle ear (impedance) and neural mechanism. Attention is called to the invariable recruitment phenomenon in every case in which nerve deafness is indicated by a loss in bone conduction. Attention is called also to the fact that at times the ear deafened from a neural lesion may appear to hear even better than the opposite, better ear. The real significance of this phenomenon is obscure. From the data set forth, it is apparent that it will require less intense masking noises to mask an ear with nerve deafness than one with impedance deafness and therefore that whenever an ear is easily masked (i. e., by a sound at or just above its threshold) it is either a normal ear (which can be easily verified) or an ear suffering from a lesion of the neural mechanism of hearing. It follows that even a faint tinnitus at or below the testing frequency will readily mask an ear with neural deafness and that the true threshold may be observed only in the absence of tinnitus. However, the ratio of recruitment of loudness furnished a means for estimating the masking effect from tinnitus. If tinnitus originates purely in the middle ear, the loudness balance shows a tendency to level up only until the tinnitus is masked by the testing frequency. After this, the loudness difference between the two ears is maintained throughout the rest of the intensity scale. If tinnitus is of purely neural origin, the loudness balance will continue to level up even after the tinnitus is masked by the testing frequency. It is impossible for a patient with hysterical deafness or a malingerer to reproduce with any approach to accuracy the tests based on loudness balance. The

binaural or monaural balance tests do not ordinarily require masking to eliminate contralateral audition, but the use of masking in conjunction with the elicitation of recruitment phenomena gives promise of interesting revelations. The recruitment phenomena are not limited in application to the neural mechanism of hearing. With appropriate stimuli they may be used to study the relative intensity of sensations from other sense organs as compared to the normal. They therefore may be used to investigate the sensations of sight, touch and pain, the static sense and so on.

EDITOR'S ABSTRACT.

LIGATION OF INTERNAL JUGULAR VEIN IN LATERAL SINUS THROMBOSIS. J. R. NOVICK, Laryngoscope **47**:325 (May) 1937.

Novick points out that one of the dreadful complications of disease of the middle ear and the mastoid is thrombosis of the lateral sinus. Removal of the clot and ligation of the internal jugular vein have been the surgical procedures in the management of this complication. Before considering the evidence in favor of and that against ligation of the internal jugular vein in a case of lateral sinus thrombosis, the author shows that thrombosis in its inception is a protective mechanism. Nature has set up its most effective barrier in the attempt to stay the invasion of the blood stream by pathogenic micro-organisms. The status, however, cannot be predetermined, and the possible sequels may be one of the following: organization, recanalization, calcification and suppurative liquefaction. After reviewing the history of the surgical management of thrombosis of the lateral sinus, the author says that in a given case the method of treatment may be one of the following: 1. Medical treatment, including (*a*) frequent small transfusions of ordinary whole blood or blood from an immunized donor; (*b*) chemotherapy, including varying drugs; (*c*) vaccines, autogenous or stock; (*d*) serums, specific or otherwise. 2. Surgical intervention in the lateral sinus; i. e., reopening of the mastoid wound, exposing and examining the lateral sinus and removing the clot or evacuating the abscess if that is found; or, if the disease is secondary to an infection of the middle ear, a simple mastoidectomy followed by exposure and examination of the lateral sinus and removal of the clot. 3. Ligation of the internal jugular vein, in addition to operation on the lateral sinus. Which of these courses one should follow will depend on several factors. Thus, if the sepsis is not much more severe than it was in the primary disease, if the temperature has not risen more than 1 or 2 degrees, if the Schilling index shows no shift or only a slight shift to the left, if the culture of the blood is negative and if the patient is not extremely prostrated, it is justified and safe to keep hands off and watch the patient carefully. In a good many such cases complete cure has resulted under the medical measures outlined above. On the other hand, if the patient is not doing well under medical treatment, it is advisable to reopen the mastoid wound or do a simple mastoidectomy if none has been done previously, and expose and examine the lateral sinus and remove the clot or drain the abscess if one is found in or around the sinus. Ligation of the internal jugular vein, however, should be resorted to only in selected cases. The author reviews case reports from the records of the Graduate Hospital of the University of Pennsylvania, which serve to illustrate the more severe type of infection. In all these cases thrombosis, accompanied by one or more of its unfavorable sequels, has complicated the clinical course of the disease. Ligation of the internal jugular vein and removal of the infected material from the sinus was resorted to. In each case this surgical manipulation has effected a rapid and almost instantaneous improvement in the condition of the patient and eventually a complete recovery. In the conclusion the author emphasizes once more that in a given case of thrombosis of the lateral sinus, definitely established with the aid of all diagnostic means, one should not act hastily. With few exceptions, this is not an emergency in which one has to rush in and ligate the jugular vein immediately. The patient should be carefully observed and treated medically as long as his or her condition permits, and just as soon as the surgeon feels that more radical measures are

essential he should reopen the mastoid wound or do a mastoidectomy if one has not been done, then plug the sinus above and below, incise it and test for bleeding from each end. Now, if there is bleeding from both ends and the surgeon is reasonably sure that the blood is coming from the sinus itself and not from its tributaries and he may assume that there is no thrombosis in any part of the sinus or the jugular vein, the sinus should be packed, the mastoid incision dressed and nothing further done. If, however, there is bleeding from below and none from above, he may assume that there is a thrombus somewhere between the upper plug and the torcular Herophili. The thrombus should be looked for and removed if possible, the sinus packed and the mastoid wound dressed. Ligation of the internal jugular vein in this instance will be of no further help, since in this area there are many other pathways for the infection to gain entrance to the systemic circulation. On the other hand, if bleeding is obtained from above and none from below, which means that a thrombus is in all probability present either in the bulb or the vein itself, then ligation of the internal jugular vein, followed by removal of the clot, is justified and should be done. EDITOR'S ABSTRACT.

PURULENT MENINGITIS: NINE CONSECUTIVE CASES WITH SEVEN RECOVERIES. J. ADAM and E. A. M. CONNAL, J. Laryng. & Otol. **52**:87 (Feb.) 1937.

This series of 9 consecutive cases of purulent meningitis complicating suppuration of the middle ear, with 7 recoveries, interested Adam and Connal because their previous experience had been one of uniform failure. An analysis of 623 cases of meningitis (other than meningococcic or tuberculous), mainly following otitis or sinusitis, by Neal, Jackson and Appelbaum showed only 16 recoveries (about 2.5 per cent), and Neumann during six years had 22 recoveries (37 per cent) in 59 cases, only 1 of the successfully treated patients being over 15 years of age. In the present series of 9 patients, 6 were over 15, of whom 5 recovered. The total recovery rate is 77 per cent, but of course 9 is far too small a number to serve as a basis for ratios. Of the patients who died, 1 was in a hopeless condition on admission, and the other would probably have been saved by better technic. Further, whereas Neumann did not open the dura, the authors have done so without exception. The experience of one of them in 2 previous cases suggested dural incision. This was done, together with regular lumbar puncture and in 5 cases with the injection of air, from 10 to 30 cc., through the lumbar needle, in accordance with Mayer's suggestion. The incision seemed helpful; certainly it has done no harm. Less cerebrospinal fluid has, on the whole, exuded than was anticipated, because the intracranial pressure thrusts the brain against the incision. The authors give brief histories of the 9 cases. EDITOR'S ABSTRACT.

LABYRINTHINE DISTURBANCES ASSOCIATED WITH TUMOR OF CEREBELLUM AND OF FOURTH VENTRICLE. M. AUBRY and J. LEREBOULLET, Ann. d'oto-laryng., January 1937, p. 1.

Aubry and Lereboullet point out that the vestibular disturbances are dependent neither on the type of tumor nor on the intracranial pressure but rather on the location of the tumor with regard to the vestibular nuclei. Since the central vestibular zone is anatomically a comparatively old portion of the vestibular tract, it has a comparatively high resistance to pathologic lesions. Thus only a rather severe lesion, that is, one in the central zone, can produce abolishment of vestibular functions. Compression of these zones, for instance by a tumor in the neighboring region, without actual destruction in the zones elicits, as a rule, only mild central disorders. However, changes in the cerebellum, which are so far away from the vestibular nuclei that a destruction or a compression is out of the question, cause practically no vestibular symptoms or only extremely mild ones.

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EDITOR'S ABSTRACT.

PHLEGMONOUS ERYSIPELAS AS SEPTIC FOCUS AFTER OPERATION ON MASTOID PROCESS. E. ROENAU, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:214, 1936.

Roenau reports the clinical histories of 5 patients. The reported cases indicate that after surgical intervention on the ear not only an ordinary wound erysipelas but a phlegmonous erysipelas of the neighboring soft parts may develop. As to how these types of phlegmon can be differentiated from erysipelas, Delbanco and Callomon emphasized that the phlegmonous infiltration is much harder than erysipelas. Moreover, the phlegmon lacks the sharp demarcation and the mirror-like surface of the erysipelas; the coloration is more bluish and the lymph nodes are more severely involved in cases of phlegmon. To be sure, these differential diagnostic factors are inadequate in some cases, and so the author adds the following criteria: 1. In phlegmonous erysipelas the cutaneous edema often appears earlier than does the reddishness, whereas in ordinary erysipelas the reverse order is the rule. 2. Phlegmon is accompanied with a rapid impairment of the general condition, even in young and strong persons. This is not true of ordinary erysipelas. 3. Petechial hemorrhages indicate sepsis and thus phlegmonous erysipelas. 4. The development of trismus and of pharyngeal phlegmon (with suppurating or serous exudate) indicates phlegmonous erysipelas, since such symptoms are never found in ordinary erysipelas. 5. Whereas ordinary erysipelas usually spreads anteriorly, in the direction of the face, phlegmonous processes have a considerable tendency to gravitate; from the ear they soon spread to the neck and to the thorax. 6. Phlegmonous erysipelas develops chiefly after operation for cholesteatoma. The author emphasizes these points not only for differential diagnosis and prognosis but for therapy, for the phlegmonous process requires radical surgical intervention.

EDITOR'S ABSTRACT.

POLYOTIA. O. KAHLER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:248, 1936.

Kahler says that the older literature contains reports of several cases of polyotia which, according to Marx, were erroneously designated as such, since in the majority of cases there was merely an auricular appendage or a double deformity. The only case of supernumerary auricle so far reported is that of Bol and de Kleyn. The case reported in this paper is that of a normally developed nursling aged 3 months, who was referred to the author for the surgical removal of a supernumerary deformed auricle. The patient was otherwise free from deformities. There was, 5 cm. above the ear, a tumor-like deformity which gave the impression of a rudimentary ear. In the center of the formation there was a small meatus, which apparently was closed at the end. A roentgenogram revealed that from this rudimentary auricle a passage led into the cranium. A roentgenographic exposure according to Schüller disclosed that this passage reached to the upper pyramidal surface. A lateral exposure indicated that it apparently went in the direction of the nasopharynx. An exposure according to Stenver revealed a cranial defect at the site of attachment of the deformity. The most noteworthy aspect was that the rudimentary ear moved during the process of swallowing. The deformity was removed during local anesthesia. A pedicle was found to enter the cranial cavity. After ligation the pedicle was cut. The cranium was found to have a circular hole 0.5 cm. in diameter. On account of severe bleeding, a contrast filling of the defect was dispensed with, and after tamponade with a muscular flap the skin was sutured and the healing was by primary intention. The skin which covered the opening did not move during the act of swallowing. The histologic study of the removed specimen disclosed that the tumor-like formation contained fat, loose and solid connective tissue, striated musculature, a mixed gland of the type found in the oral cavity, cartilage, bone and a tooth. This indicates that the growth was not a rudimentary ear and consequently the case not one of true polyotia. The author assumes that the growth developed during the early embryonal stage from the first pharyngeal pouch.

EDITOR'S ABSTRACT.

OTOGENIC MENINGITIS DUE TO GREKOWITZ BACILLUS IN TWINS. A. HEINDL JR., Monatschr. f. Ohrenh. **70**:1511 (Dec.) 1936.

Grekowitz described a gram-negative rod of the hemoglobinophilic group, which he designated as Bacterium haemophilum mucosum and which he observed in 4 cases of suppurating meningitis and 1 case of pneumonia. In 4 of the cases acute otitis existed and in 1 the pus contained the aforementioned micro-organism. Heindl observed a case of bilateral otitis with meningitis caused by the Grekowitz bacillus in a 5 month old nursling. It was the more noteworthy because a twin sister of the patient had died of the same disorder six weeks previously. After a detailed report of the clinical history and of the necropsy, the author describes the results of the histologic examination of the temporal bones, which indicated otogenic meningitis, severe acute otitis media with involvement of the medullary spaces and suppurating labyrinthitis of both sides. The route of transmission could not be definitely identified, but it was considered possible (1) that the otitis by way of the fenestrae produced labyrinthitis, which in turn produced meningitis or (2) that the invasion of the meninges took place by way of the tegmen or the edge of the petrosa. According to the serial sections, either route was possible.

EDITOR'S ABSTRACT.

OTOGENIC COMPLICATIONS OF MESOTYMPANAL PERFORATION AND SUPPURATION OF MIDDLE EAR. T. MOTLOCH, Monatschr. f. Ohrenh. **70**:1519 (Dec.) 1936.

Motloch, in reviewing the literature on complications following mesotympanal perforation, found 13 cases in which sinal complications developed, 8 cases of cerebral abscess and 1 case of meningitis with a direct route of transmission. In the other cases meningitis was accompanied with sinus thrombosis or abscess of the brain. From this the author concludes that the majority of complications after mesotympanal suppuration are caused by scars and by pathologic changes in the lymphatic system. The high incidence of abscess of the brain seems to be caused by changes in the lymph vessels. The author, however, describes a complication following mesotympanal perforation which presented the aspects of acute otitis. The patient was hospitalized with erysipelas and an acute exacerbation of chronic otitis. After the erysipelas had subsided, the ear was completely healed, but sixteen days later meningitis developed, and there was a fatal outcome. Necropsy revealed meningitis originating in the left ear. On the right side cholestaetoma was found. The examination of the cerebrospinal fluid revealed Streptococcus mucosus. The case represents the rare concurrence of mucosus otitis and mesotympanal suppuration. To what extent scar formations prevented complete cure could not be definitely determined. In view of the clinical course, it seems justificable to assume that in this case the virulence of the pathogenic organism was the cause of the complication.

EDITOR'S ABSTRACT.

DEVICE FOR PROTECTION OF EAR IN NOISY SURROUNDINGS. R. PERWITZSCHKY and LANIG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:178, 1936.

Perwitzschky and Lanig, in order to construct a device for protection against noise, analyzed the noises in railroad workshops, particularly those noises that develop during work on boilers and so on. The protecting device which they constructed on the basis of their observations consists of a metal capsule which covers the entire auricle. After describing the construction of this capsule, they describe tests of its efficacy.

EDITOR'S ABSTRACT.

Pharynx

SOME TUMORS AND ULCERS OF THE PALATE AND FAUCES. W. HOWARTH, J. Laryng. & Otol. **52**:1 (Jan.) 1937.

Howarth deals only with the more interesting tumors and ulcers of the palate and fauces, and particularly with those types which he himself was able to observe.

He disregards the small papillomas and fibromas that are usually attached to the surface of the palate or faucial pillars and begins his discussion with the type known as mixed tumors or mixed parotid tumors. The latter term is applied to them because of their close resemblance to the tumors that occasionally appear in the parotid gland. These tumors are the most common that occur in the substance of the palate. The origin of these tumors has been the subject of considerable controversy. They used to be called myxoendotheliomas, although some considered that a carcinomatous element was present in some of them. A number of different names have been applied to them, but the author thinks that it is best to retain the term "mixed tumor" on account of the varied histologic aspects. The surgical treatment is usually simple in the case of these tumors as they shell out readily, and if the entire capsule is removed cure is usually effected. After citing two interesting examples of this type of tumor, the author discusses hemangioma and hemangiofibroma of the palate. He says that these tumors are rare, but that the possibility of their existence should be considered so as to avoid mistaking them for peritonsillar abscesses. He discusses fatty tumors of the palate, which he regards as extremely rare. After mentioning a case of adenoma, he discusses adenocarcinoma and osteoma. He cites a few cases of epithelioma of the palate and fauces and discusses the operation in these cases, particularly diathermic excision. Then he gives a survey of the therapeutic results he obtained in 61 cases of malignant disease of the palate and fauces. After this, he directs attention to some forms of ulceration that are met with in the region of the palate and fauces, such as carcinomatous ulceration, the ulcerations of syphilis and tuberculosis and particularly the "precancerous epitheliomatosis." He cites 3 cases which he thinks illustrate that a precancerous condition can and does exist in the palate and fauces.

EDITOR'S ABSTRACT.

IMMEDIATE TONSILLECTOMY OR SEROTHERAPY FOR SEVERE PERITONSILLAR PHLEGMON. PREDESCU-RION, Ann. d'oto-laryng., January 1937, p. 39.

Among the 864 cases of tonsillar abscess which Predescu-Rion observed in the course of seventeen years, there were 49 in which the condition was grave, which he discusses in this report. The clinical histories of 8 cases are reported in detail. On the basis of his experiences, the author emphasizes the value of serotherapy. To be sure, satisfactory results are obtained only if sufficiently large doses of serum are given. For children he recommends 100 cc. of the antistreptococcus serum and for adults 200 cc. If necessary, this dose should be repeated on the following day. The author employed this treatment with good results in approximately 300 cases; that is, he resorted to it not only in cases of tonsillar abscess but in cases of sore throat.

EDITOR'S ABSTRACT.

SURGICAL TECHNIC OF BLEEDING NASOPHARYNGEAL FIBROMAS OF MALE PUBERTY. G. CANUYT, Ann. d'oto-laryng., March 1937, p. 203.

Canuyt points out that the bleeding nasopharyngeal fibromas that occur chiefly during male puberty are becoming rare. Rhinologists, even the directors of large clinics, often fail to encounter a single case during an entire year. The author observed a tumor of this nature in a boy aged 12 years and removed it successfully by a surgical intervention. He reports the clinical history of this patient and gives especial attention to the surgical treatment. He says that the patient was prepared for the operation by a vaccination against infection and by two blood transfusions of 100 cc. each. These preparatory blood transfusions not only stimulate hemopoiesis but exert a hemostatic action. Discussing the anesthesia, the author says that he employed local and regional anesthesia, having used this combination for many years in operations on the head and neck and particularly in extirpations of nasopharyngeal fibromas. This form of anesthesia gives great security and is satisfactory for the patient as well as for the surgeon. In regard to the surgical approach, the author says that the natural route was impractical in this case.

Moreover, although the external sublabial route, the operation of Rouge, is in favor with some rhinologists, he rejected it, because there is danger that this method of approach may be inadequate, and resorted to the transmaxillonasal approach.

EDITOR'S ABSTRACT.

TONSILS AND FOCAL INFECTION. A. MOURA, An. de oto-rino-laring. **3**:121 (June) 1937.

According to Moura, complete tonsils and those not completely removed are frequently the seat of focal infection of which infected teeth may be the suspected cause. When the tonsil is infected the symptoms intensify during tonsillitic crises, and they change after external massage on the tonsillar region. If the above changes exist, removal of the tonsils (or of the portion left after an imperfect tonsillectomy) induces recovery of the patient. Even if the symptoms are not clear, tonsillar infection may exist. Extraction of the teeth in cases of an uncertain diagnosis fails to improve the patient's condition but succeeds in discounting the theory of focal infection, which has been exaggerated.

EDITOR'S ABSTRACT.

Larynx

ABNORMAL FORMS OF TUBERCULOSIS SIMULATING CANCER OF THE LARYNX AND THEIR CONVERSE. A. HAUTANT, J. Laryng. & Otol. **52**:65 (Feb.) 1937.

Hautant shows that, in general, tuberculosis of the larynx can easily be distinguished from cancer by three principal characteristics: 1. It has numerous situations; it remains superficial, and it leaves the mobility of the vocal cord unimpaired. 2. It is accompanied by tuberculous lesions of the lung, with Koch bacilli in the sputum. 3. In histologic sections it presents giant cells, while no epitheliomatous cord can be perceived. But in exceptional cases each of these characteristics can be modified and become a cause of error. Inversely, cancer of the larynx also sometimes has the aspect of a bacillary lesion. The author considers this question worthy of extensive investigation. He limits himself to presenting a few examples, selected among the most typical of those he observed. He reaches the following conclusions: 1. Laryngeal tuberculosis can take the aspect of a warty, subglottic and unilateral lesion; sometimes it resembles a ventricular tumor. In both instances, the clinical aspect is that of an intralaryngeal epithelioma; a careful examination is necessary to avoid an error. 2. A roentgenogram of the larynx can be a valuable aid in the diagnosis of cancer. 3. When the condition is doubtful, several biopsy specimens should be taken, from different parts of the laryngeal lesion. 4. High voltage roentgen therapy, even as a test treatment, should be resorted to only with great caution. 5. Even in cases in which a diagnosis of epithelioma seems obvious, a pulmonary examination should always be made and the results taken into consideration. EDITOR'S ABSTRACT.

VOCAL DISORDERS AND VENTRICULAR CORDS. J. TARNEAUD, Ann. d'oto-laryng., April 1937, p. 314.

Tarneaud shows that the median juxtaposition of the ventricular cords serves to reenforce the sphincteral function of the larynx. By their nature, their development and their use, these superior thyroadenoid folds come into action during voluntary respiratory effort. The rapprochement of the ventricular cords permits the establishment of a strong intrathoracic pressure, such as that required for coughing. The authorities are agreed that the ventricular folds play no part in normal phonation. However, in pathologic conditions of the larynx, the ventricular cords have a function complementary to and substituting for that of the true vocal cords. They aid and replace the vocal cords in order to insure the closure

of the glottic sphincter in certain voluntary acts of respiration and phonation. The author thinks that the view of the participation of the ventricular cords in phonation deserves some correction, and he gives conclusions at which he arrived on the basis of many observations. First he discusses the voice of the ventricular folds characterized by their being in vibration. The sound is produced by them and not furnished by the vocal cords. Only the phonation produced by the vibration of the ventricular cords can be designated as the voice of the ventricular cords. Further, he gives his attention to dysphonias of the ventricular cords, which are characterized by a derangement in the function of the cords. The sound is produced by the vocal cords and changed by the ventricular cords. A whole group of dysphonias depends on the exaggerated displacement of the ventricular cords toward the median line. In the presence of a hypersthenia of this type, which is recognized by laryngologic, stroboscopic, roentgenologic and phoniatric examination, the diagnosis and the treatment are aided by local pharyngeal anesthesia. Cure cannot be obtained by symptomatic medication and requires causal treatment.

EDITOR'S ABSTRACT.

SUDDEN DEATH CAUSED BY PERFORATION OF THE TRACHEA BY A CASEOUS PARATRACHEAL GLAND. ALF GULLBRING, Nord. med. tidskr. **13**:493 (March 27) 1937.

A boy of 6 years, seven months after being treated for primary tuberculosis, localized in the right lung, fell suddenly ill while playing and complained of pains in the epigastric region and of stridor. Two hours later he died. Postmortem examination showed a hazelnut-sized gland to have penetrated into the trachea just above the right principal bronchus. The caseous matter almost completely obstructed the bronchus, and a loosened part of it had been aspirated into the left bronchus, producing atelectasis of the left lung.

WALLGREN, Göteborg. [AM. J. DIS. CHILD.]

Nose

MALIGNANT DISEASE OF THE UPPER JAW. G. ÖHNGREN, J. Laryng. & Otol. **52**:18 (Jan.) 1937.

Ohngren directs attention to the many disappointments that are encountered in the treatment of malignant tumors of the maxilloethmoid region. He says that in his clinic the surgical procedure has been abandoned in favor of endothermy, although the latter method involves great inconveniences, such as the prolonged course of healing, the objectionable odor and the tendency to late hemorrhages. The procedure in an operation by endothermy depends on the position and size of the tumor. If it is near the surface it is attacked directly with two electrodes, but if it is deep seated it must first be exposed. The author points out that since 1927 the majority of patients have been treated by a combination of surgical intervention and irradiation, the latter being applied before and after the surgical treatment. A comparison of the results obtained with irradiation and with endothermy seems to indicate that irradiation does not excel, indeed hardly equals, endothermy. In comparison with the proportion of cures achieved about ten years ago, today's results are infinitely better, but they are still far from satisfactory. More than 50 per cent of the patients treated still die within five years from the beginning of treatment, and about 20 per cent present a hopeless condition. The possibilities of achieving better results lie in earlier diagnosis and improved methods of treatment.

EDITOR'S ABSTRACT.

ANGIOMATOUS TUMOR OF NASAL FOSSAE. PIQUET and BECUWE, Ann. d'oto-laryng., May 1937, p. 393.

Piquet and Becuwe's patient, aged 32, complained of obstructed respiration and intense recurrent nasal hemorrhages, which were caused by a large angiomatous

tumor at the nasal fossae and the maxillary sinus. The authors advise resorting to Rouge-Denker's operation for access to the nasal cavities in cases of this nature. It is performed after prevous ligation of the external carotid artery, by which grave hemorrhage in the course of the operation is prevented. The author emphasizes the importance of Rouge's operation in removing the tumor, because its removal through the natural routes will be insufficient and consequently it will recur. In the author's case the satisfactory results were verified several months after the operation.

EDITOR'S ABSTRACT.

BACTERIAL ALLERGY IN PATHOGENESIS OF SPASMODIC RHINITIS. E. RICCITELLI and Y. FRANCHINI, Rev. argent. de oto-rino-laring. **6**:3 (Jan.-Feb.) 1937.

Riccitelli and Franchini point out the role of bacterial allergy secondary to tuberculous allergy in the pathogenesis of spasmodic rhinitis. The presence of tuberculous allergy can be proved by positive cutaneous and intradermal tuberculin reactions, examination of the thorax showing early tuberculosis and the presence of filtrable forms of tubercle bacilli in the sputum. Evolution of tuberculosis results in disappearance of tuberculous allergy and of the spasmodic symptoms. The authors conclude that for spasmodic rhinitis local treatments are not indicated but etiologic treatment is of value. Tuberculin therapy gives satisfactory results in controlling spasmodic rhinitis.

EDITOR'S ABSTRACT.

Miscellaneous

TWO CASES OF STREPTOCOCCIC MENINGITIS TREATED SUCCESSFULLY WITH SULFANILAMIDE AND PRONTOSIL. M. H. WEINBERG, R. R. MELLON and L. E. SHINN, J. A. M. A. **108**:1948 (June 5) 1937.

Weinberg, Mellon and Shinn used sulfanilamide and a derivative called prontosil soluble (the disodium salt of 4-sulfamidophenyl-2'-azo-7'-acetylamino-1'-hydroxynaphthalene-3', 6'-disulfonic acid) in 2 cases of streptococcic meningitis and studied its effects in comparison with those of a newly reported type of serum treatment which has been used successfully by them for nearly a decade. Of the 2 cases, the second is the one that merits the greater attention. The patient was practically moribund when she was admitted. From both the clinical and the bacteriologic standpoint the child appeared to be doomed. She had marked bulbar involvement, and the dysphagia was almost complete. "There is no reasonable doubt that a noteworthy bacteriostatic and probably a bactericidal effect of the spinal fluid is present. It is particularly clear cut in the second case, in which no growth of streptococci occurred after treatment, despite the fact that ten times the original inoculation was employed. The relative sparsity of streptococci present in the smears supported this finding." It was found that when 125 of the patient's streptococci were incubated in the patient's fresh spinal fluid, about half of them did not grow after eight hours' incubation at 37 C. After twenty-four hours none grew. Of the ultimate fate of these inhibited or killed organisms in the human being, little is known. The question of dosage and method of administration cannot be answered definitely as yet. Sulfanilamide does not seem to be toxic, so it is rather safe to use. Attempts at standardization of dosage are already being made. The authors have given normal persons as much as 4 Gm. a day, and their patients with type III pneumonia, who have shown a remarkable response (recovery in about 75 per cent), have often received as much, and sometimes more, daily. When swallowing is difficult, the drug should be given intramuscularly. The authors have overcome the difficulty of the patient's inability to swallow the tablet by administering the drug by rectum. Therapy should be started immediately. The English workers estimate that at least forty-eight hours is necessary before bacteriostatic effects can be obtained in the serum, and because of this the treatment should be instituted promptly. In their second case the authors started

the treatment in one hour and twenty minutes, the time that it took to complete the neurologic examination, the lumbar puncture and the microscopic examination of the fluid after admission of the patient to the hospital.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

MOTOR DISORDERS OF THE CENTRAL NERVOUS SYSTEM AND THEIR SIGNIFICANCE FOR SPEECH: II. CLINICAL FORMS OF MOTOR DEFECTS (THE "SPASTIC CHILD"). P. J. ZENTAY, Laryngoscope **47**:421 (June) 1937.

In this second paper Zentay deals with the various clinical forms of early motor disturbances of childhood, sufferers from which are known at present under the collective name "the spastic child." "Spastic child" is an unfortunate and confusing misnomer. It applies to, and is descriptive of, patients with only one—true, the largest—group of motor disorders of early childhood. To increase the confusion the term hardly does justice even to this group. It selects one characteristic only and ignores the many existing clinical variations. The result of the injudicious use of the term "spastic child" is that as a rule all forms of motor disorders are thrown together that actually have nothing in common with "spasticity" in the neurologic sense. It seems highly desirable that the existing chaotic terminology be clarified. Progress in this field will begin when the terminology used by the various allied specialists is brought into complete harmony with correct and up-to-date neurologic concepts. It is suggested here that the use of the all-inclusive term "spastic child" hereafter be dropped completely. Its place should be taken by the term "motor-defective child," and the condition should be known as "motor deficiency." The author classifies the central motor defects into (1) pyramidal lesions, (2) extrapyramidal lesions and (3) cerebellar lesions. His discussion is limited to motor defects that are due to antenatal or natal factors and consequently are present at the time of birth. These early disorders have particular significance for the teacher of speech, as they influence decisively the acquisition of motor functions and the development of speech. Antenatal factors are (*a*) developmental anomolies and (*b*) fetal disease of the central nervous system. Natal factors are largely represented by accidents caused by the mechanism of birth. Whatever the nature of the pathologic process is, the resultant clinical and neurologic picture may be exactly the same. However, the earlier in fetal development antenatal factors become operative, as a rule, the more decisive the defects to which they will lead. The motor defects found in a given child are not always clearcut. They may be a combination of disturbances in more than one motor system. Here again the teacher of speech will be greatly aided by correct localization, diagnosis and prognosis. After discussing the pyramidal, extrapyramidal (athetosis) and cerebellar lesions (atoxic disorders), the author reaches the following conclusions: Any motor defect must be looked on as the problem of the whole person. Motor defect in any one of the three central motor systems disturbs the tonus and balance of the other two systems also. Any motor defect interferes with the total function of the central nervous system. Any defect is irreparable, and compensation for it depends on residual functions. Improvement of functions depends largely on maturation. Any form of training must have its limitations in the potentialities of the residues. The teacher of speech must think realistically on this problem and avoid false hopes and unfounded optimism. Cooperation with the neurologist, the physical therapeutist and the psychologist is essential for best results. The total personality of the child must be evaluated. Methods of teaching and training have to be adapted not only to the nature of the motor defect but to the individual needs of the child. EDITOR'S ABSTRACT.

PATHOGENIC STAPHYLOCOCCI IN THE ANTERIOR NARES: THEIR INCIDENCE AND DIFFERENTIATION. F. A. HALLMAN, Proc. Soc. Exper. Biol. & Med. **36**:789 (June) 1937.

Cultures were made of matter from the nares of 468 persons who were without evidence of infection of the upper respiratory tract or of staphylococcic

infection elsewhere. The organisms were differentiated into pathogenic and nonpathogenic forms by means of the plasma coagulation test. In addition, cultures were obtained from 51 subjects with chronic staphylococcic osteomyelitis. Forty per cent of persons over 19 years and 60 per cent of those under 20 years who had no evidence of infection harbored pathogenic staphylococci in their nares. Among the patients with osteomyelitis the percentage of those with pathologic bacteria in the nares was slightly higher in the group over 19 years and slightly lower in the group under 20 years than in the group of normal controls. The plasma coagulation reaction was the preferred method of differentiating between pathogenic and nonpathogenic strains of staphylococci; the fermentation of mannitol, the crystal violet reaction and the production of dermonecrotoxin are also reliable confirmatory methods for such differentiation.

HANSEN, Minneapolis. [AM. J. DIS. CHILD.]

RECOVERY FROM PNEUMOCOCCAL MENINGITIS. J. R. CALDWELL and P. S. BYRNE, Brit. M. J. **1**:1204 (June 12) 1937.

A girl aged 16 had sore throat, chills and headache. There were vomiting the next day and gradual loss of consciousness. The cerebrospinal fluid was under high pressure and contained 7,000 polymorphonuclear leukocytes and extracellular diplococci in great numbers. The precipitation test was positive for the type I pneumococcus and negative for the meningococcus. Delayed cultures were sterile. Treatment consisted of repeated spinal drainage and administration intramuscularly of the disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid and by mouth of the hydrochloride of 4-sulfamido-2′,4′-diaminoazobenzene. The patient was acutely ill for only three days and had completely recovered by the eleventh day.

ECHOLS, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

NASAL SECRETION AND ENDOCRINE GLANDS. L. CHAVANNE, Ann. d'oto-laryng., May 1937, p. 401.

Chavanne concludes that clinical observations reported in the literature coincide with the results of his experiments as to the relations of the nasal secretion and endocrine glands. Castration is followed by atrophy of the glands of the nasal mucosa and lowering of the functions of secretion of the mucosa. Pathologic conditions of the nasal mucosa resulting from diminished or increased function of the genital organs, as well as transient disorders of the nasal mucosa in the course of menstruation and pregnancy, are reported. The anterior lobe of the hypophysis and the thyroid inhibit the nasal secretions. The same result is obtained by administration of extracts of the anterior lobe and the thyroid. Thyroidectomy increases nasal secretion. Thyroid extracts act through their effects on the thyroids. Extracts of the anterior lobe of the hypophysis act by contolling several endocrinotropic substances which are concerned with the nasal secretion. The author advises following a classification of angiocrine disorders of the nasal mucosa.

EDITOR'S ABSTRACT.

ANATOMIC RELATIONS OF THE CENTRAL PATHWAYS OF THE VESTIBULAR SYSTEM. P. VAN GEHUCHTEN, Rev. d'oto-neuro-opht. **15**:321 (May) 1937.

The vestibular system is isolated from the cortical and striatal centers and from the cerebellum. Its two chief functions are to maintain the reflex equilibrium of the body and to contribute to the reflex motility of the eyes. These are rendered possible by the multiple connections of the vestibular nuclei: (1) direct connections with the spinal cord by the deiterospinal bundle and crossed connections by way of the descending fibers of the posterior longitudinal bundle and (2) homolateral and heterolateral connections with the ocular nuclei. The homolateral connections are established through the vestibulomesencephalic bundle. The crossed connec-

tions are made through the ascending vestibular fibers of the posterior longitudinal bundle and consist of fibers originating in the triangular and descending vestibular nuclei. Fibers of the posterior longitudinal bundle may be traced to the red nucleus and the thalamus. This anatomic arrangement constitutes "the primary reflex arc of the vestibular system."

The vestibular system is closely associated with the cerebellum, principally the paleocerebellum. Almost all the fibers of vestibulocerebellar association end in the roof nucleus; certain of them go to other nuclei and to the cortex of the lingula, the nodulus and the flocculus. Proprioceptive impulses from the spinal cord to the cerebellar bundle reach the vermis and are reflected through fibers of the Purkinje cells to the roof nucleus. The roof nucleus represents, therefore, a cerebellovestibular center for coordination of excitations from the labyrinth and from the periphery.

In the higher vertebrates and in man close connections between the vestibular system and the striatum are important. The centripetal pathways may be represented by ascending fibers of the posterior longitudinal bundle to the thalamus and perhaps by fibers to the red nucleus and the thalamus, either directly or by the cerebellum, or even by way of the nucleus of Darkshevitch. The centrifugal connections are less well known; it is recognized that connections exist between the globus pallidus and the perirubric region.

Afferent and efferent connections between the vestibular nuclei and the cerebral cortex seem indispensable. Stimuli received in the vestibular nuclei may reach the cortex of the frontal and parietal lobes by way of the posterior longitudinal bundle and thalamus and by the central acoustic pathway. The cortical zones probably react on the vestibular centers through Türck's bundle or the temporopontile bundle, but the actual pathway is not known. It is not certain that vestibular impressions reach the frontal lobe and the motor zone, but it is logical to assume that the motor cortex may influence the vestibular centers in the exercise of voluntary motility. Such influence may be exerted by the aberrant peduncular fibers of Dejerine, which go in part to the nuclei of the posterior longitudinal bundle and perhaps in part to the triangular nucleus.

The vestibular system is integrated with the highest mechanisms that rule automatic and voluntary motility.

DENNIS, Wagon Wheel Gap, Colo. [ARCH. NEUROL. & PSYCHIAT.]

CONTRIBUTION TO THE STUDY OF THE RELATION OF MÉNIÈRE'S DISEASE TO NEUROTROPHY: TREATMENT OF MÉNIÈRE'S DISEASE BY PROCAINE HYDROCHLORIDE BLOCK. I. T. DOROCHENKO, Acta oto-laryng. **25**:262 (May-June) 1937.

Dorochenko shows that Ménière's disease, as regards both etiology and therapy, has by no means been explained. In the works of a number of authors, the most widely varying etiologic factors are adduced in connection with this disease. Proceeding from the experimental work of the school of Professor Speransky, who has drawn attention to the special role played by neurotrophy in the origin of the disease, it is necessary to consider that role fundamental. Clinical observations, to a certain extent, confirm its importance. As regards the therapy, the use of the procaine hydrochloride block proposed by A. Ubischnevsky is indicated, with the help of which in cases of Ménière's disease it is possible to rearrange the grouping of the nerve correlation, which brings about the reestablishment of the normal function. The application of the procaine hydrochloride block in several cases of Ménière's disease gave a good therapeutic result. The histories of 2 patients, which are given with detailed examination of neural status, show rapid recovery on application of the procaine hydrochloride block in both cases. No other method of treatment was employed in either case. Subsequent observations confirm the permanence of the recovery. Detailed his-

tories of the cases and the effect of the treatment establish the connection between Ménière's disease and neurotrophy. The technic of the procaine hydrochloride block is described.

EDITOR'S ABSTRACT.

CONGENITAL DEFORMITIES OF ESOPHAGUS: ROENTGENOLOGIC AND ENDOSCOPIC STUDY. P. G. GERLINGS, Acta pædiat. **19**:370, 1937.

The first portion of this paper is a textbook discussion of the various types of atresia of the esophagus. The advantages of esophagoscopic diagnosis are emphasized. In those rare cases in which the stenosis results from the presence of a thin diaphragm, the obstruction may be relieved through the endoscope.

In the second part of the paper Gerlings discusses the less common but more remediable congenital esophageal stenosis. Usually this anomaly does not produce symptoms until the patient is several months of age, commonly when solid food is added to the diet. Two cases are reported in which the condition was treated successfully by dilation through an esophagoscope. The first patient was 3 months old at the time of the onset of symptoms; the second patient vomited from birth onward but did not come under treatment until the age of 18 months.

The third anomaly discussed is congenital short esophagus. This condition was formerly regarded as rare but is now known to be fairly common. It is frequently associated with partial ectopy of the stomach. Symptomatically either dysphagia or pain may be present. The roentgenographic features are fairly characteristic; through the esophagoscope one may see dilatation of the upper portion of the esophagus with chronic esophagitis, stenosis of the esophagus, gastric mucosa just below the stenosis and in some cases peptic ulcer of the esophagus. Two cases are presented. Both patients were about a year of age; 1 showed an ulcer of the esophagus.

The fourth group of anomalies which is considered is that characterized by dilatation of the esophagus without any history which suggests the swallowing of caustics or the presence of spasm. This group is less well defined than the others because of the difficulty of excluding acquired stenosis and because of the lack of precise knowledge concerning the diameter of the esophagus in the healthy child. Examination of a series of normal infants showed that the esophagus becomes less distended by air when solid food is being swallowed than when liquids are being ingested.

MCCUNE, New York. [AM. J. DIS. CHILD.]

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

HARRIS P. MOSHER, M.D., *President, Presiding*

Seventy-First Annual Meeting, Atlantic City, N. J., May 5-6, 1938

ABSTRACTED BY DR. ALFRED LEWY, CHICAGO

(Concluded from page 838)

Friday Morning Session, May 6

REPORT OF THE BOARD OF TRUSTEES OF THE RESEARCH FUND. DR. JOHN B. RAE, New York.

The annual meeting of the trustees of the research fund was held at the headquarters of the New York Academy of Medicine on Jan. 8, 1938. The morning session was devoted to informal discussion, reports from all investigators who had received grants having been distributed to all members prior to the meeting.

Various requests for grants were considered. The increase in income was already accounted for by conditions of gift, so that no new allocations could be made without encroaching on investigations already under way. The board was therefore obliged regretfully to refuse further grants meantime, and the chairman was requested to communicate this decision to applicants for grants.

At the afternoon session Dr. Wesley C. Bowers, of New York, was presented to fill the vacancy caused by the untimely death of Dr. Arthur B. Duel.

After discussion of heredity and environment in connection with otosclerosis, the question of investigation of similar twins of known otosclerotic heredity was raised. It was hoped that the study of the onset and progress of the disease in these similar twins in varying environments would prove of value in the investigation.

It was determined to seek the aid of a geneticist as a consulting member of the board to direct the method of attacking the problem, and the chairman has since announced that Dr. Charles B. Davenport has kindly accepted the invitation and will so serve.

Dr. Wilson then read his report reviewing the work of the various investigators during the year. On motion, the thanks of the board were extended to Dr. Wilson for his able presentation.

REPORT FROM THE BOARD OF TRUSTEES OF THE RESEARCH FUND. DR. J. GORDON WILSON, Chicago.

There are wide anatomic variations in the region between the stapes and the cochlea, the site of predilection for otosclerosis.

In man, the most characteristic finding is remnants of the cartilage of the fissula ante fenestram which persist into late life and may result in otosclerotic changes.

Dr. Bast finds that the utriculoendolymphatic fold may act as a valve and close the utricular opening and cause changes in pressure.

Dr. Anson shows that there are histologic features capable of the mechanism of absorption.

Dr. Anson and Dr. Wilson find that in cases of Paget's disease the otic capsule resists the osteolytic process which destroys other parts of the temporal bone.

The air cells of the petrous portion appear as outpocketings of the mucosa, arising from the area beneath the semicanalis musculi tensoris tympani, and extend

inward, forming a loop around the carotid canal. They communicate with each other but not with the epitympanic cells of the mastoid. Their normal orifices may be the "fistulas" found at operation.

Earlier phases of the development of the otic capsule than those described by Dr. Bast have been studied, and the results will be published. The arterial and venous supply of the capsule is being studied in animals.

Thirty-four cases in which the roentgen and pathologic findings in the mastoid and petrosa have been studied by Dr. E. P. Fowler Jr. will be reported in the *American Journal of Roentgenology*. A long-standing discharge from the middle ear may be due to osteomyelitis. Study has also been made of a case of multiple myelomas of the temporal bone.

There is a division of opinion as to whether the Wever and Bray phenomenon depends on the integrity of the organ of Corti or may be due to vibration of an electrically polarized resonant structure (basilar membrane).

It is desirable to improve the tests of auditory deficiency now in use to detect the early spread of otosclerotic lesions. Roentgenography has not been satisfactory. Dr. Fowler's "hobble" effect may prove an important aid.

Dr. Knudsen and assistants at Los Angeles have an elaborate setup to investigate the question of selective amplification best suited to different types of impaired hearing.

Drs. Collet, Mortimer and Wright are studying the effect of a selected group of endocrine glands on auditory acuity.

Dr. Culler has investigated the acoustic pathway, particularly in the geniculate bodies.

The committee proposes to investigate the influence of environment on otosclerosis and has engaged Dr. Davenport as consultant.

REPORT OF THE CHICAGO MENINGITIS COMMITTEE. DR. ALFRED LEWY, Chicago.

Five cases were reviewed:

CASE 1.—A patient with hemolytic streptococcic meningitis apparently received no benefit from treatment until sulfanilamide was administered intraspinally, after which there was a prompt recovery.

CASES 2, 3, 4 and 5.—Hemolytic streptococcic meningitis occurred early in the course of otitis, without evidence of extension by way of the mastoid, and there was no surgical intervention beyond paracentesis. Sulfanilamide therapy and supportive treatment, including blood transfusion, were used. The 4 patients recovered.

Case 1 raises the question of adequate concentration of sulfanilamide in the spinal fluid. The other 4 raise the question of whether surgical intervention should not be omitted for fulminating conditions and for those of doubtful otitic origin.

REPORT OF THE REPRESENTATIVES OF THE AMERICAN OTOLOGICAL SOCIETY ON THE COMMITTEE FOR HEARING AIDS AND AUDIOMETERS OF THE COUNCIL ON PHYSICAL THERAPY OF THE AMERICAN MEDICAL ASSOCIATION. DR. HORACE NEWHART, Minneapolis.

The committee consists of nine members. Because of technical difficulties in formulating specifications, it has so far not recommended any audiometer as acceptable.

Instruments of real merit are available, equipped with bone conduction receivers, masking devices, built-in microphones and other features. To the otologist in urgent need of an audiometer it is suggested that he purchase one adapted to his need with the proviso that the manufacturer will guarantee, for a reasonable consideration, to make good by exchange or new parts whatever is necessary to meet the specifications to be adopted by the committee. It is felt that such terms are considered just and reasonable by high grade manufacturers.

The problem of a high grade receiver is not yet solved.

There is need for two types of audiometer:

(*a*) An audiometer for research and for testing the severely deaf, with complete range in pitch and intensity in a continuous band as well as selection at fixed intervals.

(*b*) A pure tone audiometer at low cost for group use, with a range of 128 double vibrations to 8192 double vibrations, not for diagnostic purposes.

Dr. Newhart presented the new Maico No. 6 school audiometer. It is designed for air conduction only, and the five octaves 128 to 8192 cycles are used.

Audiometers of this type, because of ease of operation and low cost, will find use in schools and health services.

Since there is no bone conduction or masking device, the nurse or technician is not tempted to make diagnoses.

Also, two semiportable vacuum tube hearing aids for severe nerve deafness were presented.

[In the November issue, page 777, appears an article by Dr. Newhart, entitled "A New Pure Tone Audiometer for School Use," which includes the part of this report that deals with the Maico No. 6 school audiometer.]

SYMPOSIUM: WHAT CAN BE DONE FOR CHRONIC PROGRESSIVE DEAFNESS?

A Study in the Treatment of Deafness. Dr. C. Stewart Nash, Rochester, N. Y.

I have observed 200 cases for an average of five years and ten months and wish to report those in which some improvement may be expected in the patients. The youngest patient was aged 5 years; the oldest, 78. The duration of deafness before treatment varied from a few weeks to several years. There were no controls. Various conservative methods were employed. Accurate records were kept. Four conditions are included: (1) chronic catarrhal otitis media, (2) chronic purulent otitis media, (3) nerve deafness and (4) otosclerosis.

Fifty-eight per cent of the patients have normal drum membranes; 52 per cent, open eustachian tubes; 46 per cent, partially opened tubes, and 2 per cent, tubes persistently constricted.

Treatment consisted of (*a*) correction of infective processes and (*b*) attempts to restore the conductive mechanism of the middle ear by inflation, massage, surgical intervention in the nose and throat when indicated, correction of dental malocclusion, thermal procedures, removal of the foci of infection, the giving of strychnine and other tonics, phonographic exercises, application of high frequency vibration, general treatment, endocrine therapy, administration of vitamin B and other measures.

The results indicated by the audiometer were as follows: patients with chronic catarrhal otitis media, 52 per cent better and 44 per cent worse, with chronic purulent otitis media, 47 per cent better and 47 per cent worse; with nerve deafness, 25 per cent better and 75 per cent worse; with otosclerosis, 57 per cent better and 42 per cent worse.

When the patients were tested by the spoken voice and whisper the results were essentially the same, except for those with otosclerosis, of whom 41 per cent were better and 58 per cent worse.

It may be concluded that the present treatment of chronic progressive deafness is not satisfactory.

DISCUSSION

Dr. Harris P. Mosher, Boston: No other paper has taken up this subject as fully as Dr. Nash's has. He has shown statistically what accomplishment can be expected in the treatment of chronic progressive deafness.

He feels, as I have felt for a long time, that the smugness of many otologists—and I include myself—in regard to the treatment of chronic progressive deafness is not justified.

Fifty per cent success is not so bad, if those are his statistics, but it is necessary to find something new, something that has not yet been found out, before anything satisfactory can be accomplished in the treatment of chronic progressive deafness. What that will be, I do not know.

Finding foci of infection has not been fully satisfactory in curing this condition. I have a feeling (and it is easy to go off the handle in this matter) that food allergy may play a larger part in chronic progressive deafness than is at the present time supposed.

Dr. Marvin F. Jones, New York: One of the conditions most frequently met in the office of the otologist is chronic deafness. Regardless of statistics and present knowledge of pathology, some patients feel better after inflation.

There are fluctuations which may be interpreted as improvement. Therefore, taking the lead from what Dr. Mosher has said, I believe otologists must enlist the aid of physicists and biochemists, in addition to physiologists and pathologists. The vascular system and its control, the sympathetic nervous system, may be concerned.

If such proves to be the fact and potential deafness can be diagnosed early, there is hope.

Dr. Harris H. Vail, Cincinnati: I am not satisfied with Dr. Nash's classification of chronic progressive deafness.

There is a large field for improvement in the treatment of patients with fluid in the ear who present a history of a closed ear following a cold of some months' duration for which nothing was done. When seen by an otologist, the ear is in the stage of adhesions and sclerosis. If a paracentesis had been promptly done, the chances are that the ear would have fully recovered.

There is one method which may be of help, galvanism, and there are several parasympathetic stimulants, vasodilators. I mention one, mecholyl, which can be put into the ear by galvanism. I do not think it is indicated in cases of otosclerosis.

Dr. J. Gordon Wilson, Chicago: More knowledge of progressive deafness is desirable, and Dr. Nash's statistics are of value to supplement the knowledge concerning this condition and to suggest intelligent treatment. Especially is this so when one must deal with cases in which two factors are present—heredity and present or past environmental disease. Otosclerosis is a well known example of a condition in which disease elsewhere can play a part, but proof is wanting.

I have seen a few cases of otosclerosis in which after an attack of allergy the hearing was permanently diminished.

Data are not available regarding what environmental diseases act as causes of occurrence and progress. When ankylosis of the stapes has set in, present available methods are hopeless to effect a cure.

Dr. James A. Babbitt, Philadelphia: Some months ago I became interested in Grant Selfridge's work on vitamin deficiency and nutritional rearrangement.

With others, I began experimenting with a combination of vitamins B and G in connection with liver and iron. We obtained dramatic results. They may or may not be permanent, but I think here is a line of research which ought to be followed.

Dr. F. R. Spencer, Boulder, Colo.: I have had 3 patients for whom correction of faulty basal metabolism has improved a minor defect in hearing.

The mother of 1 had chronic progressive deafness of high degree.

It seems that investigation of the endocrine system is worthy of more consideration.

Dr. Gordon Berry, Worcester, Mass.: This condition tends to become less progressive in patients between the ages of 25 and 50. Roentgen, electrical or endocrine therapy may reactivate progressive deafness that has become dormant.

If one can hold what one has, that is some gain. In an effort to help, one must be sure not to aggravate the loss.

Dr. E. P. Fowler, New York: It is a mistake not to put down the age of the patient, as the results of treatment differ much according to age.

One of the main functions of the otologist should be prevention of deafness. Many types may be prevented if the patient is seen early. A continuing plea for prevention is worth while

Dr. C. Stewart Nash, Rochester, N. Y.: Dr. Jones' work on allergy in relation to the middle ear must be considered. Dr. Vail's suggestion that paracentesis is beneficial in the treatment of serous otitis media is correct. It was impossible to include all the various pathologic conditions producing deafness; hence this condition was included under the heading of catarrh.

Dr. Fowler's point that age is important and Dr. Wilson's point on vascularity, allergy and heredity as factors are matters on which I agree. I also have used vitamins B and G as has Dr. Babbitt but have not formed any opinion as yet.

Dr. Spencer's opinion concerning basal metabolism fits in with my experience.

Dr. Berry's plea for early recognition prompts me to say it would be a great consolation to know what to do.

Rationale, Technic, Case Reports and Observations of Grafts in the Round Window. Dr. Walter Hughson, Abington, Pa.

An operation designed to place a graft of tissue in the niche of the round window has already been reported as a possible treatment for deafness due to nonsuppurative disease of the ear. This procedure has now been done 25 times, and in 2 cases both ears have been operated on. An analysis of the audiometric records has been made to determine changes in the hearing during the actual operative period, or the time during which the drum remained open; the immediate response following closure of the drum, and the late effect of the procedure on subjective and objective response. In 2 cases improvement has not resulted. In all other cases which have been observed for three months after operation, improvement has developed for certain frequencies. Improvement is approximately 10 decibels for these frequencies, with an extreme of 35 decibels. Subjective improvement is accepted only on the basis of new sounds heard and greater ease in understanding speech. Otoscopic observations are included in the analyses of cases.

The rationale of the procedure in the light of the new surgical procedures on the labyrinth for the treatment of deafness becomes somewhat more difficult of elucidation. However, from observed and recorded facts, fixation of the membrane of the round window with a tissue graft does have an effect on the hearing of deafened persons which cannot be explained simply on the basis of myringotomy. Improvement has been continuous for a period of twenty months, the longest period of observation in the series. Particular pains have been taken to eliminate remote factors which might have been responsible for the loss of hearing before undertaking any surgical treatment.

The results obtained would seem to warrant further employment of the method in selected cases of impairment of hearing.

DISCUSSION

Dr. D. E. Staunton Wishart, Toronto, Canada: Dr. Hughson's is the last of a series of four papers; the first two dealt with experimental data; the last two, with results on patients.

There is no adequate statement of the kind of deafness for which the operation is recommended. The audiometer tests are given, but the essential point is ability to hear the spoken voice. Also in the report of the experimental test the cochlear response (Wever-Bray) cannot be taken to represent the actual hearing.

Of 4 photomicrographs of the round window in animals after operation, 2 show an air space between the graft and the membrane of the round window. It is

much more difficult to insert a graft in the human angled niche than in the direct bay of the cat.

Dr. Hughson has submitted a detailed account of 8 cases. In the present paper he adds 18 more.

Dr. Hughson's technic is to be highly praised, but the result he claims, 10 decibels' improvement in the critical frequencies, when analyzed on the basis of his own charts, may be disputed, and for the present I do not believe the results warrant the risks of operation.

These criticisms are founded on a theoretic consideration of Dr. Hughson's own papers.

For the manner of conduct and the report of his procedure Dr. Hughson is to be highly complimented, and, while I do not believe the operation will be successful when performed by others, further elucidation of the problem could not be left in better hands than Dr. Hughson's.

Dr. Walter Hughson, Abington, Pa.: I regret that Dr. Wishart's prepared discussion refers to early experimental findings and a preliminary report rather than to the present paper. The facts reported this morning are a matter of record and require no theoretic interpretation.

The operation is applicable in any case of conduction deafness in a nonsuppurating ear with intact drum and a tympanic cavity not obliterated.

The operation was based on animal experiment, under rigid controls. Acoustic stimulation was found increased immediately for higher frequencies. The response in human beings does not coincide. Cochlear response in animals is not necessarily an index of subjective hearing.

All my patients had been treated, sometimes over long periods, without benefit. In no instance was one made worse. There were no infections.

Subjective improvement is treacherous ground. No case was reported until improvement became a matter of course rather than the sensational experience of the moment.

The photomimeographs studied by Dr. Wishart were made from animals ten days to seven weeks after operation. Unfortunately for this discussion I cannot demonstrate the oldest graft, twenty months after operation. I did not state that these grafts increase intralabyrinthine pressure. According to a suggestion offered by Dr. Pohlman, fixing the round window increases the reflecting surface of the cochlea.

The composite audiograms present a definite average improvement. I am not familiar with any comparable results from any form of therapy.

Most deaf persons have been made critical by years of futile treatment. I am able to give them reasonable assurance of substantial improvement in properly selected cases.

Operation on the Nasopharynx in the Treatment of Chronic Progressive Deafness. Dr. Harold Walker, Boston.

Chronic progressive deafness may be divided into two classes, (1) otosclerosis and (2) progressive loss of hearing due to nasopharyngeal and other infections. It is important that the cause be diagnosed. Otosclerosis may exist without recognition, owing to obstructive changes in the middle ear caused by infection. The important factors in the second class of cases are prevention of infective foci before marked deafness occurs and eradication of these foci when they are present.

Normal nasal breathing is the important factor in the prevention of infections in the nose and throat, which are the primary causes of chronic progressive deafness. In order to obtain this condition there must be an elimination of all nasal and pharyngeal obstructions which prevent the free passage of air through the middle meatus and the nasopharynx.

DISCUSSION

Dr. Joseph C. Beck, Chicago: An important note of the paper is that one should have a good nose.

Recently I prepared a chapter for Dr. Fowler on the eustachian tube and learned some things regarding its importance in a consideration of progressive deafness. A new note in connection with the eustachian tube is the change in the angle of the jaw in the mandibular joint. Of interest also is Dr. Walker's statement in connection with the condition of air on the rooms. It would be well if installation of air conditioning could be expected soon throughout all homes, so that the preparation of the room for the patient would not make so much difference.

In regard to intranasal operative conditions, it is generally known that good results are obtained early. Later it does not matter. I have operated on many septums in all these years but have not seen any improvement in hearing. Deafness is not an indication for operation on the septum. In 1 case, that of a deaf-mute, excellent results were obtained by opening the eustachian tube.

Dr. Max Goldstein, St. Louis: I do not recognize such a thing as a "deaf-mute." Such a person is congenitally deaf or totally deafened but has the potentiality of speech.

It is an interesting observation, however, that when adenoidectomy was at the height of its popularity almost every school for the deaf was filled with children not congenitally deaf who had large adenoids and subsequent adhesions and sealing of the whole eustachian tube. The entire conduction mechanism and the nerve as well were put out of commission. The cochlea finally became involved, whatever its physiologic function might be.

Dr. F. R. Spencer, Boulder, Colo.: In 1929 Dr. Sidney Yankauer emphasized the importance of infections of the pharyngeal bursa in increasing the impairment of hearing. These must be seen by direct nasopharyngoscopic examination.

Dr. Walker mentioned the importance of this fact. I have used this nasopharyngoscope for many years. He mentioned the importance also of opening the bursa with a probe.

After adenoidectomy remnants of lymphoid tissue may remain. In Rosenmueller's fossa they do more damage than over the posterior wall.

I have used lipoiodine, as recommended by Lillie. Perhaps an iodide would do, but I continue to remove these islands with the cautery or by surgical diathermy.

Dr. D. Harold Walker, Boston: In all the cases there is lowered bone conduction, especially for sounds in the high range. Why is this?

I am not referring to the eustachian tubes; they are affected only by a secondary infection.

My theory is that if the patient breathes normally, through his nose, there will be fewer sinal and pharyngeal infections and less absorption. I have not referred to infected teeth or other foci, which, of course, are important.

For deaf children, I suggest that the congenitally closed eustachian tube be treated as I have outlined.

Friday Afternoon Session, May 6

Tuning Fork Tests Reported in Terms of Decibels. Dr. Robert J. Hunter, Philadelphia.

I shall describe a method by which the decrement of a fork may be measured by ear and the results given in terms of decibels. No knowledge of logarithims is required to get all other data about a fork if the significance of the numbers 4.34, 0.6931 and 3.0103 is understood. With these numbers any fork tests can be given in terms of decibels and it is advisable that forks be standardized in this way. Tuning forks vary in decrement and cannot be precise. A good audiometer is an instrument of precision.

DISCUSSION

Harvey Fletcher, Ph.D., New York: Dr. Hunter's picture is too pessimistic. The type of tuning fork used was affected by the way it was held or activated, and this influenced his results.

He then demonstrated by slide a mathematical method of determining the decrement, which does not lend itself to abstracting.

At the international conference last summer, it was agreed that decrements be expressed in decibels. I hope otologists will have their forks calibrated in decibels per second. Then the loss of hearing can be determined in decibels by subtracting the time in seconds during which the fork is heard from the normal hearing time and multiplying the remainder by the constant of the fork expressed in decibels per second.

DR. A. A. HAYDEN, Chicago: The work of physicists, such as Dr. Fletcher and Dr. Hunter, is important in the application of the physical principles underlying the construction of audiometers, but I believe that otologists are no more interested in the details of the logarithmic formulas and so on that have been shown this afternoon than the listener to an orchestra is interested in finger exercises. They are interested only in the final results. The welfare of the patient is the final answer.

DR. ROBERT J. HUNTER, Philadelphia: The point I wanted to make is that one may forget the symbols and simply divide by the numbers. I realize this is not scientific, but if otologists will think in terms of decibels and are given a method whereby they can do this easily a step in advance will be made.

The forks used were intentionally ordinary ones, not the Bezold Edelman forks. They are too expensive.

Although theoretically the amplitude diminishes regularly, forks vary according to manufacture, the way they are activated and held and other factors.

Therefore, I feel that the audiometer is a more nearly standard and accurate measure than tuning forks, but a number do not have audiometers, and they may use this technic.

USE OF THRESHOLD AND OF LOUDER SOUNDS IN CLINICAL DIAGNOSIS AND THE PRESCRIBING OF HEARING AIDS. DR. EDMUND P. FOWLER, New York.

New methods for accurately determining the threshold for bone conduction and for measuring tinnitus and its effect on obstructive and neural deafness will be presented.

I recommend any audiometer which meets and maintains the minimum standards set by the Committee on Physical Therapy of the American Medical Association, if manufactured by a responsible firm. Methods involving complicated charts should be avoided.

By the use of accurate methods, new phenomena have been discovered. One of these, "recruitment of loudness with intensity," differentiates nerve deafness from obstructive deafness.

Fundamental Observations.—The Logan scale is logical. This is the musical scale. Hearing curves plotted on the sensation scale are more satisfactory for most purposes. The lower tone limit is 20 double vibrations; the upper, between 20,000 and 25,000 double vibrations. The older tests were faulty because the effect of intensity was not measured. An audiogram form in which a straight line represents the average normal curve is advocated. A dependable diagram must record both air and bone conduction at several frequencies. Loss of hearing for high tones in nerve deafness and for low tones in obstructive deafness is not universal. The opposite ear must be masked if the threshold is down 45 decibels. A soundproof room is necessary for dependable measurement of the threshold, but for prescribing hearing aids conversation in average normal surroundings is better.

Timber.—Tone quality probably varies in reception by different brains. If a band of frequencies is missing, the sound ear can substitute, or even the diseased ear can, to some extent, supply the missing frequency and also restore the sense of loudness because of the pick-up on both sides of the gap.

Testing at the Threshold.—The response is clearcut in cases of nerve deafness but not so clear in those of obstructive deafness. This is due to the "recruitment"

phenomenon demonstrated by throwing alternately into each ear increasing intensities and balancing the sense of loudness binaurally. This test is becoming increasingly useful.

A Test for the Accuracy of Determinations of Bone Conduction.—In the absence of nerve deafness, air conduction will balance with bone conduction binaurally with equal increments of loudness.

A case is presented in which 45 decibels at 256 double vibrations and 35 decibels at 128 double vibrations above bone conduction are required. The threshold to balance in loudness is 5 decibels above the air conduction threshold. This indicates nerve deafness affecting low tones.

Measurement of Tinnitus and Its Effect on the Hearing.—The loudness of the tinnitus should be matched against the loudness of similar frequencies (or frequency) in the opposite ear. The number of decibels above the threshold in the ear without tinnitus required to balance the loudness of the tinnitus is the measure of the loudness of the tinnitus. If the number of decibels representing the loudness of the tinnitus is substracted from the total neural deafness, the remainder will be the actual loss of neural hearing in decibels.

Hearing Aids.—The first requisite is to bring to the ear a level of sound adequate for good hearing and agreeable to the hearer. The ear should receive from 15 to 50 decibels above the minimum audibility of speech, according to distance and surrounding noise. In cases of pure obstructive deafness simple amplification is sufficient. In cases of nerve deafness the problem is more difficult, because levels of loudness are influenced by the recruitment phenomenon, overloudness must be controlled according to the ratio of recruitment and there must be also selective repression, for instance of low sounds. This is not possible with a light portable hearing aid.

Nearby undesirable sounds or many voices nearby mask the speech one desires to hear.

In cases of obstructive deafness this is sometimes an advantage, as the speaker automatically raises his voice, but the hearing aid does not distinguish between desirable and undesirable sounds.

Some kind of control should be devised to prevent sound over a certain intensity, which may cause damage, from reaching the ear.

New Phenomenon.—Some sounds may sound louder to a deaf person than to one with better hearing. Lorente de Nó's researches supply three facts to explain this recruitment of loudness: 1. Stimulation of a neuron requires that several impulses impinge on it in less than 500 microseconds. 2. The frequency of impulses conducted by a nerve fiber cannot exceed a certain characteristic for each fiber (approximately 800 per second). 3. The strength of stimulation is proportional to the number of impulses per unit of time. Consequently, when a group of neurons are missing, sufficient loudness will bring the remaining ones into activity and deliver to the higher centers as many impulses as from the normal side. This accounts for full recruitment.

Overrecruitment indicates that with sounds beyond a certain intensity a larger number of impulses reach the centers from the diseased than from the normal side. Transmission of impulses through the primary acoustic nuclei is interrupted by phases of depressed excitability. This phenomenon is similar to the "inhibition of the spinal flexor reflex." If the organ of Corti is diseased, the inhibitory mechanisms may when overloaded allow a larger number of impulses than normal to reach the cortex. The tone seems louder but is perceived as a mixed tone or even merely a noise. This is what happens in "overrecruitment of loudness."

HARVEY FLETCHER, PH.D., New York: Dr. Fowler is to be complimented on the new concepts presented. I was especially interested in his suggestion for measuring tinnitus and his method for determining the true threshold for bone

conduction. He had the patient balance the tone perceived by bone and by air conduction at various levels above the threshold, thereby eliminating tactile sense as a source of error.

The validity of bone conduction tests at low frequencies has long been questioned. Dr. Fowler has offered a method of determining the true threshold for bone conduction.

DR. MAX GOLDSTEIN, St. Louis: For the more serious types of deafness, for instance in children, one needs an audiometer regulated to include much higher intensities than do many of the instruments now in the field.

The low limit of 128 double vibrations is not enough. At the beginning of conduction deafness, the patient may hear 128 double vibrations, but one does not know if he hears 64. I have been urging the committee to include this requirement.

After all, the audiometer is not merely an instrument of measurement for a hearing aid; it is intended as a diagnostic instrument, and for this purpose it should have a tone frequency from 64 to 8192 double vibrations and beyond.

DR. A. A. HAYDEN, Chicago: Dr. Fowler first interested the Western Electric Company in audiometers in 1919, but even now the man who invented the audiometer and the man who makes it do not speak the same language. That has been one of the decided drawbacks in the way of general acceptance of this instrument, which in my opinion is needed by otologists and hard of hearing people.

The type of instrument designed by Dr. Goldstein, with a broader range, may be necessary for the Central Institute for the Deaf, but for the otologist's office it is better to have an audiometer that is not prohibitive in price. Establishing requirements which will make the instrument too expensive is the very thing that the consultant otologists of the committee of the Council on Physical Therapy of the American Medical Association have tried to avoid.

DR. EDMUND P. FOWLER, New York: Perhaps I can make myself clearer. If the bone conduction is normal, one should put in the bone conduction sound 10 decibels above the threshold and balance it against a sound by air conduction 10 decibels above the threshold. If the thresholds are true, they should come near the same loudness at 10 decibels above either by bone or by air conduction.

In this and similar cases, although the air conduction goes up 5 to 10 decibels correctly, the patient does not hear the increase in bone conduction until it reaches 40 decibels. For this patient it took that degree above the threshold of bone conduction to balance a 5 decibel increase by air conduction. His condition is not really obstructive deafness; there was not perfect appreciation by the nerve at these frequencies.

HARVEY FLETCHER, PH.D., New York: I doubt the findings.

DR. EDMUND P. FOWLER, New York: The patient is given air conduction alternating with bone conduction. It is just as definite as if a person weighed two things in his two hands and compared them.

DR. MOSES H. LURIE, Boston: Are bone conduction and air conduction measured from the same zero line?

DR. EDMUND P. FOWLER, New York: In regard to low frequencies, Dr. Fletcher and I have said that 128 (also 256) double vibrations will give all the information lower frequencies will give if the tests are accurately made and applied.

I regret that Dr. Lorente de Nó and Dr. Steinberg were not here to discuss measurement of tinnitus, as the thoughts set forth here have many interesting implications.

XANTHOMATOSIS (SCHÜLLER-CHRISTIAN DISEASE): REPORT OF A CASE IN WHICH THERE WAS A RADIOSENSITIVE PATHOLOGIC GROWTH IN THE MASTOID. DR. JOHN J. SHEA, Memphis, Tenn.

The pathologic structure of xanthomatosis is radiosensitive and is due to a disturbance of the lipid metabolism. Perivascular infiltration occurs, producing a degeneration of the parts affected.

Report of Case.—A white youth aged 16 complained of vertigo and loss of hearing in the right ear. Recent orthopedic examination had resulted in a diagnosis of fusiform cyst of the femur. The Wassermann and Kahn reactions were negative and the Meinicke reaction, 2 plus. Since roentgen therapy was effective in the treatment of the cyst, it was applied to the temporal bone and was successful. Subsequently, polydypsia, polyuria and general weakness developed. Roentgenologic study revealed cystic degeneration of both lungs. Insufflation of solution of posterior pituitary was successful in controlling the diabetic symptoms. Reexamination at Johns Hopkins Hospital confirmed the diagnosis. Surgical treatment of these lesions leaves a discharging wound which fails to heal.

Conclusions.—The pathologic structure of Schüller-Christian disease is radiosensitive. Further study is necessary to discover a means of readjusting the disturbed lipoid metabolism. Surgical intervention should be avoided.

DISCUSSION

Dr. LeRoy A. Schall, Boston: Five clinical entities are recognized as xanthomatosis, namely, Gaucher's disease, Niemann-Pick disease, Schüller-Christian disease, the xanthomas occurring in icterus, diabetes and pregnancy and the so-called essential xanthomatosis.

Schüller-Christian disease is of otologic interest. It is characterized by granulomatous deposits on the bones of the skull. In 12 cases studied, 3 patients showed involvement of the mastoid. They were treated by a diet low in fat and by irradiation. Study of a case at the Massachusetts General Hospital indicated that the result of roentgen therapy is nonspecific fibrosis. When the condition is associated with diabetes insipidus, solution of posterior pituitary is indicated.

Dr. V. V. Wood, St. Louis: I agree with Dr. Shea and Dr. Schall as to the futility of operation, and I should like to report a case of three months' duration, in which there were bilateral discharging postauricular swellings and a fatty swelling over the left triceps muscle.

New bone lesions can form rapidly. In my case roentgenograms showed that decalcification in the skull, the right and left ribs and the right tibia had occurred in three months despite roentgen therapy.

Therapeutically, roentgen rays, solution of posterior pituitary, a diet high in vitamins, heliotherapy, injections of foreign proteins and blood transfusions were ineffective.

Autopsy showed destruction of the entire mastoid, which had been replaced by fatty tissue, and the fingers could be inserted into the brain.

The case has been completely reported in the *Annals of Otolaryngology, Rhinology and Laryngology* (**46**:991 [Dec.] 1937).

Report on the Result of Submitting Dogs to Rotation. Dr. D. R. Arellano, Havana, Cuba.

Dogs were delabyrinthized on the left side, bandaged like a mummy and placed on a revolving platform in such positions that in turn the right horizontal, the right anterior vertical and the right posterior vertical canal were in the plane of rotation, in each case counterclockwise and then clockwise, so that each experiment had six stages. The rotations were carried out at various speeds for twenty seconds each time.

(The procedure was illustrated by lantern slides.)

Findings.—The canals of mammals, unlike those of lower vertebrate animals, may be stimulated by movement in either direction. Each canal is more easily excited by rotation in one direction than by rotation in the opposite direction. The horizontal canal responds best to an ampullipetal, and the anterior vertical to an ampullifugal and the posterior vertical to an ampullipetal movement.

Time does not allow descriptions of other interesting results, which will be published in full.

DISCUSSION

PROF. JOHN TAIT, Montreal, Canada: I wish to make a correction. This important work was done not under my supervision but independently by Dr. Arellano and Dr. Abalo, working in Cuba.

They began some years ago, and the many films, only a part of theirs, indicate their industry and expenditures. Dr. Arellano has elected to present his method and only briefly his results, drawn from large numerical data.

With one labyrinth removed, the dog has three single canals. At any selected speed of rotation any single canal is responsible for a certain number of nystagmic beats. The numbers differ as they are counted during or after rotation. They also differ according as the rotation has occurred in the ampulla-leading (ampullifugal) or in the ampulla-trailing (ampullipetal) sense.

The averages are as follows:

	Ampullifugal	Ampullipetal
Horizontal	17	24
Anterior vertical	16	3
Posterior vertical	5½	8

Each canal has its preferential sense: the horizontal is ampulla trailing, the anterior vertical ampulla leading, as found by Ewald in the bird, and the posterior ampulla trailing. This result for mammals is entirely new and is perfectly dependable in the case of the dog.

In normal dogs with both labyrinths functioning the investigators obtained a greater number of beats for each rotation. By comparison they obtained a percentage ratio of activity of the individual canals, as follows:

	Ampulla-Leading	Ampulla-Trailing
Horizontal	43	60
Anterior vertical	57	11
Posterior vertical	19	28

The percentages for the horizontal canal total 103, but those for the anterior with the posterior of the opposite side are short 15 per cent. Adding 19 and 11 gives 30 per cent, which leaves a greater shortage.

Dr. McNally and I think the utricular maculas make a separate contribution to the number of nystagmic beats.

Two human subjects were tested by Dr. Arellano and Dr. Abalo, but results were omitted from the paper.

If nystagmic figures are obtained in a sufficient number of cases it will be possible to diagnose a lesion of a single canal.

THE ENDOLYMPH. DR. LEROY M. POLVOGT, Baltimore.

The perilymph communicates with the subarachnoid space, but the endolymph is in a closed system and comprises only a few drops. It is of importance in the functioning of the vestibular and the auditory apparatus. No blood vessels have been demonstrated in the end organs of the cochlear nerve, and it is believed that its nutrition is brought and its waste products carried off by the endolymph.

Guild's experiments indicate that the stria vascularis secretes the endolymph. Lesions of the stria do not necessarily impair the hearing. Results of chemical examination should be correlated with those of hearing tests. The direction of flow is from the cochlear and the vestibular system through the ductus endolymphaticus to the saccus endolymphaticus and probably through its walls to the venous system.

Dr. Crowe has recently shown that physical or chemical disorders of the endolymph may be responsible for Ménière's disease. In this disorder the lesion is an irritative one, not destructive, since there are long remissions, and the symptoms may fluctuate. The primary cause may lie in the endolymph.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM AMICITIÆ SACRUM

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.
Place: Brussels, Belgium. Time: September 1939.

HUNGARIAN OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. Treer, Bocskay-u. 9, Szeged.
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SOCIEDAD RIOPLATENSE DE OTO-RHINO-LARINGOLOGÍA (ARGENTINE SECTION)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

SOCIÉTÉ FRANÇAISE D'OTO-RHINO-LARYNGOLOGIE

Secretary: Dr. Henri Flurin, 19 avenue Mac-Mahon, Paris (17°).
Time: Oct. 24-26, 1938.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: St. Louis. Time: May 15-19, 1939.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha
Place: Chicago. Time: Oct. 8-14, 1939.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. John D. Kernan Jr., 120 E. 75th St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. George B. Wood, 504 Physicians' Bldg., Philadelphia.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Drake Hotel, Chicago. Time: May 9-11, 1939.

SECTIONS:

Eastern.—Chairman: Dr. Frank E. Kittredge, Masonic Temple, Nashua, N. H.
Place: Boston. Time: Jan. 11, 1939.
Southern.—Chairman: Dr. Francis E. LeJeune, Maison Blanche Bldg., New Orleans.
Place: New Orleans. Time: Jan. 14, 1939.
Middle.—Chairman: Dr. T. R. Gittins, Davidson Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: Jan. 19-20, 1939.
Western.—Chairman: Dr. Frederic G. Sprowl, Medical Arts Bldg., Spokane, Wash.
Place: Spokane, Wash. Time: Jan. 29, 1939.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Isidore Friesner, 36 E. 73d St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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